The Manitoba Medical Review

Vol. 40 APRIL. 1960 No. 4

Medicine

A Clinical Aid in Differential Diagnosis of Dyspnoea "The Valsalva Manoeuvre"

B. H. Lyons

Two hundred years ago Valsalva[1] described a method of expelling pus from the middle ear by having the patient perform a prolonged forceful expiration against resistance. One hundred years later, Weber[2] noted that the pulse became small in volume during this procedure. This observation suggested an explanation for a phenomenon known from ancient times, that prolonged forceful expiration could cause syncope. It is only in the last two decades however, that physiological observations and experiments demonstrated that complex changes in pulse and blood pressure occur during this manoeuvre. The methods of study required intra-arterial punctures to record pulse and pressure changes with simultaneous cardiac catheterization, circulation time measurements and other methods of examination.

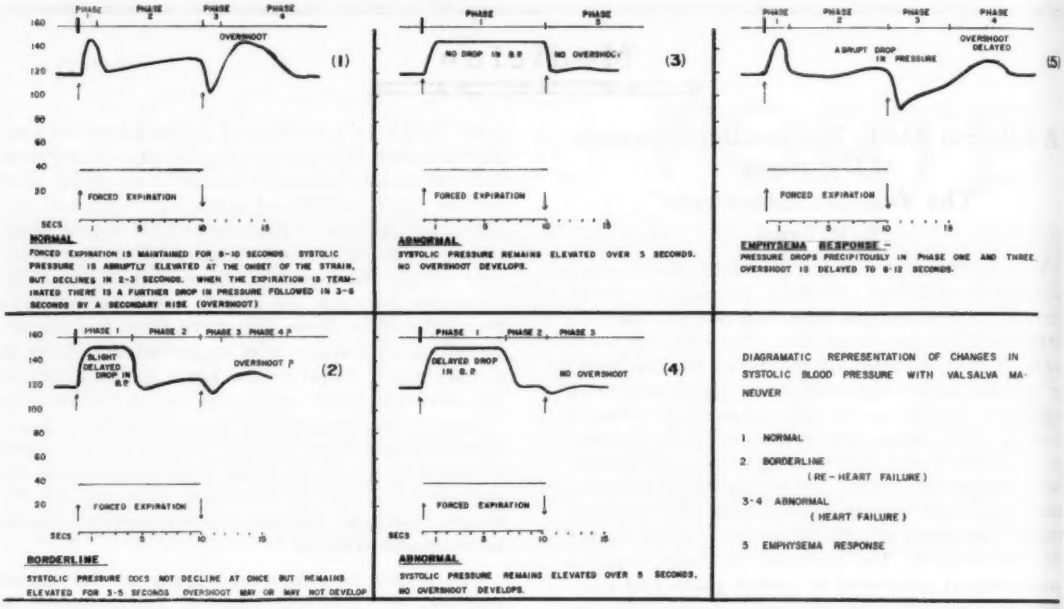
When a forced expiration is maintained for 8-10 seconds, a predictable series of pulse and pressure changes develop in the normal individual. The moment the forced expiration is begun, there is an abrupt elevation of systolic pressure (usually 20-40 mms.), which is maintained for 2-3 seconds. This rise is attributed to sudden increase in intrathoracic pressure producing a forceful expulsion of blood from heart and lungs into the systemic arteries, and is designated as "Phase 1." Since the elevated intrathoracic pressure presents an impediment to venous return from the great veins into the thorax and heart, cardiac output falls. Systolic pressure drops to approximately resting levels and pulse pressure narrows. Now there may follow a slow rise in pressure until the end of the forced expiration. This period is designated as "Phase 2." When the forced straining is discontinued, there is a sudden drop in pressure for a period of 3-6 seconds ("Phase 3"). During this phase, previously dammed up venous blood below the diaphragm has flooded into the thorax and therefore there now follows an increased cardiac output with a secondary rise in pulse and blood pressure of 20-30 mms. for a few seconds. Bradycardia is usually associated. This secondary rise in "Phase 4," has also been called the "Overshoot." In some normals the overshoot may fail to appear.

In 1944, Hamilton[3] discovered that there was considerable difference in the responses of patients with circulatory failure, but it has only been since 1952, that a number of investigators[4-13] have demonstrated these differences clearly. They found that the systolic pressure instead of dropping after two or three seconds of straining, remained elevated for at least 5 seconds and in severe cases for the total duration (8-10 seconds) of the expiratory effort. Furthermore, there was little or no "overshoot" in the last phase. Explanations have been offered for these variations from the normal. In left heart failure, it is suggested that there is a reservoir of blood in the lungs which increases the available blood to be expelled at the onset of straining, thus prolonging the arterial rise. When there is right heart failure, there is increased pressure in the great veins which overcomes the opposing intrathoracic pressure generated by the forced expiration and maintains a supply of blood to heart and arteries.

The above interpretations apply to the supine position. In Fowler's position, improvement in borderline abnormal tests may occur.

Because intra-arterial tracings were required to record these findings, the studies remained essentially laboratory procedures, of limited practical application. This limitation was overcome by Knowles et al[9] who described a simple bedside method using an ordinary sphygmomanometer instead of intra-arterial tracings to record pressure changes. While the information obtained is not as complete or precise as by the latter method, it nevertheless is sufficiently accurate to enable conclusions to be reached in most cases. Knowles' observations have since been confirmed by others.

The method in detail as applied by the writer follows: The nurse first acquaints the patient with the procedure and has him practice it two or three times. The tubing of a blood pressure apparatus is attached to a mouthpiece. The subject takes a moderate inspiration and then expires forcefully into the tubing to produce an elevation of 40 mm. mercury in one second. He maintains pressure at this level for a period of 8-10 seconds. Leakage around the lips is avoided. At the nurse's signal, the forced expiration is abruptly discontinued. The 10 seconds' time is preferred, but 8 seconds is used in patients who have difficulty carrying out the test, and in those with severe emphysema, or coronary disease. In emphysema of severe degree, prolonged expiration may induce syncope, and in coronary disease, angina has been reported. It is of interest that angina has not been recorded during the test if heart failure co-existed. This indicates that the anginal pain is induced by the abrupt



drop in blood pressure, which of course occurs only if there is no decompensation.

When the nurse has completed her instructions to the patient, the physician proceeds with the test. With a second sphygmomanometer he records the systolic pressure several times to get a baseline. He then inflates the bulb to elevate the mercury to 40 mm. above the basal systolic pressure. The physician now signals and the patient expires forcefully into the other sphygmomanometer. Usually the doctor will at once hear blood pressure sounds. If he does not hear them, he halts the test and tries again, elevating the pressure only 20-30 mm., which nearly always is successful. Normally, he will hear two or three beats, and then silence as the pressure drops. The time elapsed before the pressure drops is recorded, either by a signal to the nurse who is holding a stop watch, or by counting the beats heard and correcting for the pulse rate. For the remainder of the forced expiration (during the second and third phase), the doctor may try to follow the pressure down, but this is somewhat difficult. In most cases it will suffice, if he prepares to catch the overshoot (phase 4). To do this he sets the pressure to 15 to 20 degrees above the baseline and notes the time from the termination of the Valsalva effort to the reappearance of sounds.

The test is usually performed in both the supine and Fowler positions; doubtful cases will show a better performance in Fowler's.

In carrying out the test, as has been indicated, an experienced nurse or assistant is desirable. Satisfactory readings can be obtained in first trials in perhaps 75% of patients examined. Often a reliable test is obtained only on a repeat visit when the co-operation of the patient is improved. Failures will occur in patients who lack intelligence, who are very feeble or in some neurotics who claim they cannot maintain the expiration because they are "too weak" or "need air." (Actually patients in moderately severe failure can usually carry out the test without trouble). In the writer's experience, most failures to obtain a satisfactory result occur in patients whose blood pressure is too labile to register a steady baseline, or where there is much beat to beat variation, as in some cases of atrial fibrillation.

How reliable is this procedure? Most authorities agree that, providing the test is performed correctly, a clear cut positive or negative result correctly indicates the presence or absence of decompensation. However, if mitral or aortic stenosis is present, some state that a positive test does not necessarily mean failure, but indicates a "tight valve" as the mechanism which impedes the entry of blood during phase 2 into the arterial system. This of course is valuable information — in fact some authors recommend the test in assisting selection of patients for cardiac surgery.

Intermediate or borderline results (pressure maintained 3-5 sec.; presence of small overshoot),

indicate the need for further observation and testing. In the author's experience, most of these borderline tests have been found to be due to incipient decompensation.

As the test may require time and patience on the part of the physician, it would not be of much practical use unless it was helpful in solving difficult or problem cases. The writer has found it frequently helpful in just such situations. He has used it principally in evaluating the complaint of dyspnoea in patients who have a cardiac lesion, but whose symptoms of breathlessness could be due to an associated disorder such as obesity, neurosis, emphysema, or pulmonary sepsis. As all physicians know, the patient who has both a cardiac lesion **and** pulmonary disease, frequently presents a difficult problem, despite the assistance of laboratory examinations such as radiology, electrocardiography, circulation time and pulmonary function study. The differential diagnosis is important since the approach to therapy is different. The writer has on a number of occasions found the Valsalva a valuable additional method of examination in reaching a decision as to the etiology of dyspnoea.

Since emphysema constitutes one of the major differential diagnostic problems, it is a fortunate circumstance that in uncomplicated emphysema the results of the tests are usually clear cut, because they are the opposite of those found in heart failure — a sort of exaggerated normal response. This has been well described by Mills[12]. In phase 2 and 3, the systolic and pulse pressure drop to lower levels than in normals. For this reason the test in severe emphysema should be limited to 8 seconds, to avoid occasional syncope, (this is a mechanism of "couph syncope" which may occur in patients with lung disease after fits of coughing). After the termination of the expiratory effort the onset of the rebound is delayed, — average 12 seconds compared to half this time for normals and cardiacs. This long delay is a specific reaction for emphysema. At times, the rebound is not registered at all. This latter need not cause confusion with the lack of rebound in heart failure cases, since in decompensation the rebound failure is secondary to an abnormally maintained elevation of blood pressure during the strain, whereas with emphysema there is an early abrupt drop of pressure.

Illustrative Cases

Mrs. N. — Age 31, had rheumatic heart disease with aortic insufficiency. She was a very nervous female, complaining frequently of dizziness, submammary pains, and shortness of breath. There was never any objective evidence of failure and the symptoms were typical of a neurosis.

On January 6, 1959, she complained of increasing shortness of breath, and claimed she was now using 4 pillows at night instead of 2. Failure was suspected but physical examination and X-Ray were not revealing. A Valsalva test however was positive. She was therefore treated with rest, digitalis and diuretics. On January 13, she reported some improvement but the Valsalva was still positive. At the next visit (February 10th) she stated she was much better. The Valsalva test was now negative and remained so. The clinical course in this case confirmed the correctness of the Valsalva response.

Mr. X — Age 60, suffered from shortness of breath for many years. He had quite marked emphysema to which his dyspnoea was attributed. Also present were the ausculatory signs of mitral stenosis. Except for slight atrial enlargement the cardiac silhouette was normal. The E.C.G. showed right axis deviation and the P waves had the countour both of P mitrale and P pumonale.

He began complaining of acute nocturnal attacks of dyspnoea and cough while out of the city at the beach. It was thought at first that these represented asthmatic attacks. A Valsalva test performed when there were no symptoms was positive in the supine position. This indicated a "tight" mitral stenosis. Subsequently, he was seen during an attack. Vascular engorgement with acute pulmonary oedema was found, proving that mitral stenosis was the cause of his attacks. He later had left cardiac catheterization and a mitral commissurotomy was performed.

Mr. B. — Age 55, in 1954, had a myocardial infarct in an Eastern city. He was in and out of hospital for nine months for treatment of "heart failure." On examination in Winnipeg he complained of dyspnoea. His heart was grossly enlarged and bilateral pulmonary crepitations were heard. E.C.G. indicated previous infarction.

Treatment was continued with digitalis and diuretics, but the symptoms and findings remained unchanged. At a subsequent office visit it was noticed that he coughed frequently, bringing up greenish sputum. On questioning, he stated that this symptom had recurred periodically for many years. The question then arose whether his pulmonary findings and dyspnoea might not in reality be due to pulmonary sepsis, especially as a circulation time was found to be normal. He was instructed to stop smoking, given some expectorant mixtures with resulting improvement. In January, 1956, the writer became acquainted with the Valsalva method as described by Knowles, and this was carried out in Mr. B. and found to be normal. The result of this test together with the normal circulation time confirmed the suspicion that pulmonary crepitations and dyspnoea were due to pulmonary sepsis rather than cardiac disease. He was treated more extensively with bronchodilators and posturization and considerable further improvement was noted.

Subsequently the patient developed episodes of acute coronary insufficiency with pulmonary oedema. With these attacks, the Valsalva became

positive; with improvement the test tended to an intermediate and at times negative reaction.

The tests were of value in this case because:

1. The suspicion that pulmonary disease rather than co-existing cardiac disease was the cause of the rales and dyspnoea, was confirmed.

2. The test became positive whenever heart failure entered the picture.

Summary

The Valsalva manoeuvre is a forced expiration against resistance. Predictable changes in blood pressure occur during the test in healthy individuals. This normal response is altered in congestive heart failure and in "tight" mitral and aortic stenosis. In emphysema there is an "exaggerated normal" reaction. The method is applicable in office practice and may assist in the differential diagnosis of dyspnea. It is particularly helpful in assessment of symptoms when cardiac and uncomplicated pulmonary disease co-exist, since the responses are diametrically opposite in these conditions. If the test is negative, then the dyspnea can be attributed to the lung condition. If it is positive then heart failure is present.

References

1. Valsalva, A. W.: Quoted in New England Journal of Medicine, 253/822, 1955.
2. Weber, E. F.: Ibid.
3. Hamilton, W. F., Woodbury, R. A. and Harper, H. T.: A.J. of Phys., 141/42, 1944.
4. Goldberg, H., Elisberg, E. T. and Katz, L. N.: Circulation, 5/38, 1952.
5. Elisberg, E., Signian, E., Milles, G. and Katz, L. N.: Circulation, 7/880, 1953.
6. Greene, D. G., Bunnell, I. L.: Circulation, 8/264, 1953.
7. McIntosh, H. D., Burnam, J. F., Hickam, J. B. and Warren, J. V.: Circulation, 9/811, 1954.
8. Judson, W. E., Hatcher, J. D. and Wilknis, R.: Circulation, 11/889, 1955.
9. Knowles, J. H., Gorlith, I. R. and Storey, C. K.: J.A.M.A., 160/44, 1956.
10. Burroughs, Robert W., and Bruce, and Robert A.: Circulation, 14/72, 1956.
11. Gorlin, R., Knowles, J. H. and Storey, C. F.: A.J. of M., 22/197, 1937.
12. Mills, H. Kattus, A. A.: Circulation, 17/65, 1958.
13. Irvin, C. W.: J.A.M.A., 170/787, 1959.

A Review of Some Basic Concepts of Heart Failure

David H. Stein, M.D., F.R.C.P. (C)

In this paper an attempt will be made to review some of the basic concepts regarding the hemodynamics and treatment of heart failure. Emphasis will be placed on clinical applications of these concepts.

The fundamental lesion in heart failure is a biochemical one about which little is known. In order to define and understand the problems of heart failure, we will have to learn more about the biochemical processes which occur within the myocardial cells. Answers will have to be sought for such questions as:

1. What is muscle fatigue biochemically?
2. How and why does digitalis affect the intracellular processes of the failing heart?

Despite our lack of knowledge of the biochemical defects in heart failure, certain mechanical or hemodynamic alterations occur which are observable and measurable. Right and left heart catheterizations have made possible, with little risk to the patient, direct measurements of cardiac output and intracardiac pressures. In the past decade, much information has been acquired about the hemodynamics of normal and diseased hearts. Measurements of cardiac output and intracardiac pressures have been carried out on patients at rest, during exercise, and after the administration of different drugs [3 - 6].

From the mechanical viewpoint, all heart failure is characterized by a decrease in the pumping ability of one or both ventricles. When the myocardium becomes weak, inadequate ventricular emptying during systole occurs. This results in a rise in the diastolic volume and pressure. At first the individual myocardial fibres are able to respond to this diastolic stretching or lengthening by an increased release of energy in the following systolic contraction. This results in increased stroke volume (amount of blood ejected from either ventricle during one systole). Eventually, the myocardium may become so weakened that a rise in filling pressure is unable to elicit an increase in stroke volume. The fall in stroke volume may be compensated by an increased heart rate for a time, but a fall in cardiac output finally results. At this stage failure is clinically evident.

In the early stages of myocardial inadequacy, the cardiac output is maintained because the ventricles operate at an increased diastolic filling pressure. This is probably a good example of Starling's law of the heart. This law relates the energy liberated during systolic contraction of the ventricles to the myocardial fibre length at the end of diastole. Many criticisms can be made of Starling's law in its application to normal hearts[1, 2]. However, in the case of the failing heart, the principles of Starling's law appear quite valid.

Since the failing heart operates at a considerably increased diastolic volume and pressure, it will be an enlarged heart. This first clinical corollary may seem quite elementary. However, a substantial number of patients can be found who are under treatment for heart failure, having normal sized hearts. The rule of the enlarged heart in failure applies to the vast majority of patients with heart disease, regardless of etiology. Two possible exceptions to this rule should be mentioned. In Cor pulmonale there is usually "pure" right ventricular enlargement, which may not produce very marked cardiac enlargement on the standard P-A chest

x-ray. Constrictive pericarditis may also present manifestations of heart failure without significant cardiomegaly. This is a relatively rare condition.

Left and Right Ventricular Failure

An extremely useful concept, which clarifies much of the clinical picture of heart failure, is to consider the circulation as being comprised of two systems in series, each with its own ventricular pump.

Either ventricle may fail independently of the other and produce distinct clinical syndromes. Since the two circulations are continuous with each other, severe failure of either ventricle will cause a general reduction of blood flow.

Before considering the hemodynamics of left and right ventricular failure, some of the features of the pulmonary and systemic circulations should be reviewed.

1. The level of atrial pressure, right or left, is mainly dependent on the ventricular diastolic pressure. When the latter is raised, blood flow from the atrium involved will decrease until its distention causes the pressure to rise above the ventricular diastolic pressure when flow through the valve will be resumed.

2. The left atrium and pulmonary veins are not as distensible as the right atrium and vena cavae. A significant increase in volume of the left atrium will be reflected in a prompt pressure rise in that chamber and the pulmonary veins.

3. Since there are no valves in the pulmonary veins, any rise of pressure in the left atrium will cause a pressure rise in the pulmonary capillaries. The normally low pressure in the pulmonary capillaries (10 mm. Hg.) allows a wide margin of safety. Only when the pulmonary capillary pressure exceeds 30 mm. Hg will pulmonary edema result.

4. The pulmonary circuit comprised of the right ventricle and the pulmonary arteries, capillaries and veins, normally is a low pressure system. As can be seen from Table 1, the pressures are approximately one-fifth of those in the systemic circulation. Consequently, right ventricular work is much less than that of the left ventricle.

Table 1
Normal Pressure in the Systemic and Pulmonary Circulations

Systemic:	
Left ventricle	120/0 mm. Hg.
Aorta	120/80 mm. Hg.
Pulmonary:	
Right Ventricle	25/0 mm. Hg.
Pulmonary Artery	25/10 mm. Hg.

5. The pulmonary vascular system is an extremely distensible one, being able to accommodate two to three times normal blood flow without a significant pressure rise. In comparison, systemic arterial pressure rises promptly with slight increase in cardiac output (i.e., during exercise).

Left ventricular failure is the commonest form of of failure in patients with acquired heart disease. Either because of myocardial weakness (i.e., chronic ischemic changes due to coronary artery disease), or an excessive load (i.e., arterial hypertension or aortic valve disease), the left ventricle fails to eject during systole all the blood that is delivered during diastole. This causes a rise in the ventricular diastolic pressure. Immediately, the pressure must rise in the left atrium, pulmonary veins and capillaries. If this pressure "behind" the left ventricle becomes sufficiently high, frank pulmonary edema ensues.

The signs and symptoms of left ventricular failure relate to the lungs. The lungs are congested with blood, causing them to become stiff or turgid. This increases the work of ventilation and causes, even in the absence of frank pulmonary edema, a sensation of dyspnoea, which may be present only on exertion or in acute attacks of cardiac asthma occurring while the patient is in a horizontal position sleeping. As the failure progresses, dyspnoea may be present at rest.

Left ventricular failure is generally episodic. Between these bouts, the patient may be comfortable when at rest and may have "dry" lung fields. Nevertheless, in the presence of an enlarged heart (particularly an enlarged left ventricle), a history of dyspnoea on less than moderate exertion, or of paroxysmal nocturnal dyspnoea, is a definite indication for digitalizing the patient. Since the bouts of failure are relatively acute, there is no time for compensatory renal mechanisms which increase blood volume to take place. Therefore digitalis, not diuretics, is the treatment of choice in such patients.

The increased pulmonary blood volume and pressures which result from left ventricular failure cause an increase in right ventricular work. The latter chamber must eject its blood against an increased resistance. If such conditions are sustained, right ventricular failure may ensue. It is of interest that mitral stenosis also may produce marked pulmonary congestion and hypertension—just as is found in left ventricular failure. However, since there is, in the case of the valvular lesion, a mechanical obstruction to blood flow, therapy aimed at increasing left ventricular contractility is usually of little value.

Right Ventricular Failure

Isolated right ventricular failure is relatively uncommon in acquired heart disease. Pulmonary hypertension of various etiologies (i.e., mitral stenosis, chronic left ventricular failure, severe emphysema and other chronic pulmonary disorders) causes most of the instances of right ventricular failure.

The clinical picture of right ventricular failure is the one commonly referred to as "congestive" heart failure because of the hypervolemia and organ congestion which is usually present. The high diastolic pressure in the failing right ventricle causes a rise in pressure in the right atrium and

vena cavae. Enlarged neck veins, hepatomegaly and dependent edema are the well known clinical features of right ventricular failure. It should be stressed that all these features must be present in order to establish the diagnosis. Not uncommonly, patients are treated for heart failure solely on the basis of ankle edema which is a common phenomenon usually related to local venous disorders.

Right ventricular failure usually develops slowly. In some manner, not clearly understood, the failing heart signals the kidney to reabsorb more sodium and water, producing the hypervolemia of congestive failure. There is some controversy about whether these changes in renal tubular function are mediated by an adrenocortical hormone and/or antidiuretic hormone.

It should be pointed out that the rate of evolution of the symptoms of congestive failure does not correlate as well with the degree of myocardial inadequacy as with the dietary sodium intake.

Low and High Output Failure

At this time consideration of the terms "low output" and "high output" failure might be of clinical value. There is no physiologic reason for classifying patients in heart failure on the basis of their cardiac output. However, such differentiation may have important therapeutic implications.

There is no absolute level of cardiac output that one can correlate with the presence or absence of heart failure. The heart must pump enough blood to supply properly all the body tissues. This amount of blood depends on physiologic (exercise, etc.) or pathophysiologic (anemia, thyrotoxicosis, etc.) demands.

The large majority of patients with heart failure due to myocardial weakness and/or increased pressure loads will have a cardiac output less than normal. This has been demonstrated by various hemodynamic studies. As a result of the decreased cardiac output certain signs and symptoms are present in addition to the features of left and/or right ventricular failure. These signs and symptoms are outlined in Table 2.

Much less commonly, as a result of pathophysiological influences such as severe anemia, thyrotoxicosis, arteriovenous fistula, Paget's Disease, beriberi, blood flow is usually high even at rest. These disorders probably have the same effect on the circulation, namely, a reduction in peripheral resistance. Since the myocardium is usually adequate in the above listed pathological states, the heart is able to pump the large quantities of blood demanded of it.

However, should the heart become unable to meet these excessive demands, failure results. This failure is usually of the right ventricular or combined type. The level of cardiac output at which the failure occurs may still be considerably greater than the average normal resting output. These patients with "high output" failure have certain distinguishing features as listed in Table 2. It is extremely important to recognize these features, as almost invariably they point to some underlying non-cardiac disorder, usually curable. Often, the failure state cannot be improved until the underlying disorder such as thyrotoxicosis or anemia is corrected.

Table 2
A Comparison of Certain Clinical Features of High and Low Output Failure

Low Output Failure:	High Output Failure:
(1) Small Pulse.	(1) Full Pulse.
(2) Narrow Pulse Pressure.	(2) Widened Pulse Pressure.
(3) Extremities Cold.	(3) Extremities Warm.
(4) Pale Complexion.	(4) Often Ruddy Complexion.
(5) Faint Heart Sounds and "Quiet" Precordium by Palpation.	(5) Loud Heart Sounds and Increased Precordial "Activity" by Palpation.

Heart Rate and Failure

Very commonly, heart failure, particularly left ventricular failure is precipitated by a rapid heart rate. Since the cardiac output is a function of both the heart rate and the stroke volume, a slight rise in rate usually results in an increased cardiac output. Further increases in rate begin to shorten the ventricular diastolic filling period. Also the time required for "recharging" of the contractile proteins in the myocardial cells after each systole, may be encroached upon. Eventually, for all hearts, normal as well as diseased, there is a critical rate beyond which the cardiac output falls, producing failure. For the normal heart, this critical rate may be between 160 - 180/minute. In a patient with a reduced myocardial reserve as a result of coronary artery disease or increased pressure loads, the critical rate may be between 100 - 125/minute. Thus, very frequently acute pulmonary edema is precipitated by tachycardia secondary to respiratory infections, post-operative pain or bladder distention, emotional upsets, etc.

On the basis of the above, prophylactic preoperative digitalization is probably indicated in patients who have enlarged hearts or who have a history of long standing hypertension, coronary artery disease or aortic valvular disease. This does not imply that digitalis will slow a sinus tachycardia prior to the development of heart failure. The drug may, however, become effective in increasing myocardial contractility at the point where the cardiac output begins to fall despite a rising diastolic filling pressure. Previous objections to the use of digitalis in patients without failure were based on the belief that cardiac output was thereby lowered. Recent hemodynamic studies on normals have shown that digitalis produces no appreciable change in cardiac output or intracardiac pressure[6].

Digitalis and Diuretics

In the treatment of heart failure, digitalis preparations and various diuretic agents have become universally accepted. It is with the effects and interrelations of these two types of drugs that the final portion of this paper is concerned.

As was mentioned at the outset, the specific biochemical action of digitalis is not known. It is currently believed that digitalis affects the permeability of the myocardial cell membrane to the potassium ion. This influences the ionic environment at the contractile proteins, actin and myosin, and their phosphate energy system. During heart failure, the intracellular potassium of the myocardium is thought to be decreased. Therapeutic doses of digitalis cause a return of potassium into these cells, whereas toxic doses of digitalis reportedly increase the potassium loss from the myocardium[7].

The hemodynamic effects of digitalis are more easily studied and have been demonstrated in numerous experiments on patients with and without failure. The ability of the drug to increase myocardial contractility and thereby increase the cardiac output is of prime importance in the treatment of heart failure. Ventricular diastolic pressures and volumes decrease as the ventricular systole becomes more effective. Consequently, atrial pressures and the failure manifestations also diminish.

Digitalis has another property, of considerable importance to us, namely its ability to depress atrio-ventricular conduction. This makes possible the slowing of the ventricular response in auricular tachycardias and arrhythmias whether or not failure is present.

Toxic doses of digitalis often result in marked myocardial irritability which is probably related to the potassium shift out of the myocardial cells. This irritability may be expressed as premature ventricular beats, often coupled with the normal beats to produce a bigeminal rhythm. Another more serious product of digitalis intoxication, ventricular tachycardia, may occur, frequently with fatal results. This marked myocardial irritability is often decreased or abolished by the intravenous administration of potassium salts. In other words, digitalis and potassium have opposing effects on myocardial irritability. Potassium salts therefore are often used in the treatment of certain of the manifestations of digitalis intoxication.

Most of the diuretics currently used, whether they be parenteral mercurials or the popular oral chlorothiazide preparations, result in an appreciable loss of potassium, in addition to sodium and chloride, in the urine. If the patient develops hypopotassemia, the heart becomes excessively sensitive to digitalis. Therefore, even the routinely used daily maintenance doses of digitalis may result in serious digitalis intoxication.

Generally, patients in heart failure treated with diuretics will not develop hypopotassemia or other significant electrolyte disturbances if they are eating reasonably well. However, if for any reason such patients are unable to eat or lose potassium through diarrhoea or vomiting, digitalis-intoxication may appear rapidly.

Once digitalis intoxication occurs, this state may persist for several weeks if the intracellular potassium is not restored (there is little or no correlation with serum potassium levels). The continued use of diuretics, enhancing potassium loss, is fraught with danger when arrhythmias due to digitalis intoxication are present.

Therefore, several rules are suggested with regard to the concurrent administration of digitalis and diuretics:

1. Frequent observation of patients who are on daily digitalis and diuretic therapy. In some instances daily or weekly examinations are indicated.
2. Preferably, the use of intermittent rather than daily diuretic dosage, in order to allow correction of electrolyte loss.
3. Patients should be warned to stop the diuretic agent in the presence of vomiting, diarrhoea or marked anorexia.
4. Patients suspected of being in a state of digitalis intoxication should not be given diuretic agents of any kind.
5. If the physician is uncertain as to whether or not digitalis intoxication is present, the safest course is to stop digitalis and diuretics and observe the patient closely.
6. Physicians using digitalis preparations should be familiar with the various manifestations of intoxication with this drug. It should be stressed that 25-30% of patients with digitalis intoxication may present with serious disturbances of heart rhythm and conduction only[8]. This is particularly true of those patients receiving the purified glycosides of digitalis. The absence of vomiting, nausea, anorexia, coloured vision, etc., makes this type of intoxication insidious and extremely dangerous. We must be aware of such possibilities when prescribing digitalis, particularly when diuretics are also administered.

Summary

Certain fundamental principles governing the mechanics of heart failure have been reviewed. An attempt has been made to correlate this information with the clinical features as well as the treatment of heart failure.

References

1. Sarnoff, S.: "Myocardial Contractility as Described by Ventricular Function Curves." Physiol. Revs., 35: 107, 1955.
2. Hamilton, W. F.: "Role of Starling's Concept in Regulation of Normal Circulation." Physiol. Revs., 35: 161, 1955.
3. Hickam, J. B. and Cargill, W. H.: "Effect of Exercise on Cardiac Output and Pulmonary Arterial Pressure in Normal Persons and Patients With Cardiovascular Disease and Pulmonary Emphysema." J. Clin. Invest., 27: 10, 1948.
4. Harvey, R. M., Ferrer, M. J., Cathcart, R. T. and Alexander, J. A.: "Some Effects of Digoxin on the Heart and Circulation in Man." Circulation, 5: 366, 1951.
5. Harvey, R. M., Ferrer, M. J., Samet, P., Bader, R. A., Bader, M. E., Cournand, A., Richards, D. W.: "Mechanical and Myocardial Factors in Rheumatic Heart Disease." Circulation, 11: 531, 1955.
6. Dresdale, D. T., Yuceoglu, Y. Z., Michtom, R. J., Schultz, M. and Lunger, M.: "Effects of Lanatoside Con Cardiovascular Hemodynamics." Am. J. Cardiol., 4: 88, 1959.
7. Staub, H.: "Pharmacology of Cardiac Glycosides." Am. J. Cardiol., 3: 776, 1959.
8. Von Capeller, D., Copeland, G. D. and Stern, T. N.: "Digitalic Intoxication." Ann. Int. Med., 50: 869, 1959.

Gastroenterological Gleanings 1959

P. K. Tisdale

Department of Internal Medicine, Faculty of Medicine, University of Manitoba.
Department of Internal Medicine, Misericordia Hospital, Winnipeg, Manitoba.

The student of Gastroenterology enjoys a considerable variety in the sizes, shapes and natures of the fields in which he may glean. The purist may restrict himself to the purely systemic publications. The student with, possibly, broader interests may search the columns of publications which cover a broader field, but still may remain within the confines generally attributed to internal Medicine. There was a day when the non-specialized "Association" journals were also a fruitful source of information in a given field, but it would seem that the attraction of the specialist journals to the present day medical author and the volume of current medical literature have combined to postpone the appearance of important papers in the more general, "Association" journals of new and interesting work to somewhat later date. This very phenomenon may be utilized by the gleaner to his advantage depending upon the nature of gleanings for which he may be searching. The recent advances publicized in the "Non specialized" journals may usually be presumed to have withstood a more prolonged and rigorous "test of time" than the earlier and sometimes prematurely published "Recent Advance" which may initially be published unverified in a specialty journal.

Surveillance of a fairly representative group of publications has not revealed the occurrence of any major "Breakthrough" in 1959 in any of the major problems occupying the attention of investigators and clinicians interested in the field of Gastroenterology. Much material of interest has, however, appeared during 1959 and, in fact, much more than can be summarized or even listed in one short article. The following therefore represents gleanings which have been of particular interest to this reviewer and which seem to him to be of potential interest to some, if not all, engaged in the clinical practice of medicine.

It may be of passing interest to note that in two general journals not restricted to Gastroenterology the ratio of articles on Gastroenterological subjects to other topics was a fairly constant ratio of approximately one article in five. This ratio was established by the screening of three hundred and fifty-two articles, of which seventy were found to be concerned with Gastroenterological subjects.

Articles concerned with liver and biliary tract, peptic ulcer and gastrointestinal haemorrhage (including gastric carcinoma), and articles concerned with other regions of the alimentary canal each comprise about one-quarter of the articles reviewed. The remaining twenty-five per cent covers the rest of the field of Gastroenterology such as pancreatic disease, unusual tumours, infections, and parasitic infestations. It should be borne in mind, however, that the existence of the journal, "Diabetes" undoubtedly markedly reduces the volume of the literature on diabetes and related subjects in the periodicals reviewed. This journal was, for reasons of space, ignored for the purpose of this review.

Peptic Ulcer Disease Including Gastro-Intestinal Haemorrhage and Gastric Cancer

There has been increasing liaison between internists and pediatricians in two areas of recent years. These areas of overlap are of interest to all practicing clinicians. Firstly, in that previously rare disease, Fibrocystic Disease of the Pancreas where modern antibiotic agents and increased understanding of problems of absorption have led to the survival of these unfortunate children to adult age. Secondly, the increasingly frequent registration of the diagnosis of peptic ulcer in infants and children has also been a controversial area. Some of us have been at times sceptical of some of the higher reported incidence of this disease in infancy and childhood. The continued reporting of carefully investigated series has, however, led some of us at least to take a more charitable view of this diagnosis in infancy and childhood in recent years. Doctors Muggia and Spiro of Yale University School of Medicine review a series of twenty-four such young patients under the age of sixteen years and accompany this report with an interesting discussion of childhood peptic ulcer[1]. One can not help but agree with these authors when they suggest that the most important single factor in establishing the diagnosis is for the clinician to entertain the possibility of such a diagnosis in this younger age group. The other clinical point which catches the eye in this article is that, while the history is frequently atypical in the younger age group below the age of two years, the presenting manifestation is very likely to be an acute event such as bleeding, penetration or perforation.

The "vigorous diagnostic approach" to upper gastro-intestinal haemorrhage as advocated by Dr. Eddy Palmer, is discussed and the application of this principle to eighty-five patients, apparently consecutive admissions, is reported by Dr. Norman Scott, Jr.[2]. The reader is briefly reminded that the major feature of the "vigorous diagnostic approach" is the employment of emergency oesophagoscopy preceded immediately by an ice-water gastric lavage via an Ewald tube and usually followed up by gastroscopy if the oesophagoscopy reveals a normal gullet. The above endoscopys are then followed by barium contrast upper gastro-intestinal x-ray. The author is an enthusiastic advocate of the procedure largely on the grounds that in eleven patients (12.9% of the total) the employment of the above procedures showed the bleeding to be from "such unexpected sources that treatment directed at the suspected cause would have been totally ineffective or even harmful." The most important

region of confusion seems to be the distinction between lower oesophageal origin of the haemorrhage and gastric or duodenal origin. It is difficult to be very severely critical of an author who has the courage of his convictions and furthermore comes up with a fairly respectable mortality figure. One cannot agree with him outright, however. There are two thoughts that come to mind as one peruses this article, firstly that the author was spared the embarrassment of inserting his Ewald tube or his oesophagoscope into an aneurysm of the thoracic aorta. Routine employment of this vigorous approach would have led this reviewer into such an unfortunate predicament on at least two occasions in the last fifteen years. It was in fact the narrow escape from such an accident that led this reviewer to adopt a more conservative attitude toward endoscopy in major haematemesis. The author also cites one of his fatalities as having been due to necrosis of the duodenal stump occurring in a patient who underwent gastrectomy to control major persisting and continuing haemorrhage. One wonders whether this patient would have required emergency surgical intervention if he had not been subjected to the "vigorous approach" technique. This article does, however, give one occasion to pause and consider that possibly one is too conservative in his indications for endoscopy in haemorrhage.

Two unusual sources of significant gastrointestinal bleeding are reported and though neither are necessarily new they are both of interest and worth jogging our memories for. Bralow and Girsh[3] report a patient suffering from massive haematemesis following oral penicillin anaphylaxis. The patient was admitted in anaphylactic shock following the ingestion of a single tablet of oral penicillin. Haematemesis was occurring, but initial haemoglobin was 18.6 grams obviously with a haematocrit of 55% representing haemoconcentration. Lowest level of haemoglobin recorded was 13.8 grams. Subsequent to response of allergic manifestations to adrenalin and antihistamins, barium studies of stomach showed a rigidity interpreted as probably being carcinomatous. Twenty-four hours later repeat barium studies of stomach were normal. Gastroscopy subsequently revealed thickened hyperemic redundant gastric mucosa with the appearance of sub-mucosal infiltration involving almost the entire wall of the stomach. Twelve days later gastroscopy showed a normal distensible stomach with a mild superficial gastritis. The authors also point out that the objective findings of allergic gastric-urticaria as observed by x-ray and gastroscope may persist for two weeks after specific allergic therapy has been instituted and may be confused with carcinoma. Serial studies should show progressive diminution of the lesion to the vanishing point in these patients.

The question of "stress ulcers" and "stress" duodenitis is discussed by Albert M. Katz[4]. He describes a series of autopsies on patients dying in shock chiefly from myocardial infarction (25 cases were collected in six years of autopsy. Total number of autopsies during this period not stated). Twenty of the 25 died of acute myocardial infarction. All showed "haemorrhagic duodenitis of more than minimal degree." It is suggested by the author that the mechanism of this "haemorrhagic duodenitis is comparable to that postulated in acute "stress" ulcers and that this process probably accounts for a good deal of the coffee grounds and occasional bloody emesis which we observe early in severe myocardial infarction patients.

Experimental phenylbutazone ulcer in dogs is reported by Varro, Cserney and Javor[5]. They demonstrate, apparently satisfactorily, that phenylbutazone ulcers in dogs are associated with disorders of secretion of both acid and pepsin, and they presume therefore that the drug affects both the chief and parietal cells. It is of interest that, though the work reported in this article is experimental work on dogs, these ulcers exhibit two of the characteristics clinically observed in humans, namely the "explosiveness" of the occurrence of the lesions—the dogs were gastroscoped daily, and ulcers would be seen full-blown in an area which the previous day had been completely normal. The other clinical characteristic was the strong tendency to bleeding—77% of the observed ulcers in these dogs bled. The low gastric acidity associated with these lesions suggests that the mechanism of etiology differs from that of human peptic ulcer, which is again in agreement with the clinically observed facts that phenylbutazone ulcer in humans is not restricted to individuals who have previously exhibited the diathesis nor is it observed to significantly herald the onset of a recurrent classic peptic ulceration. The authors do not postulate an etiologic mechanism.

Ulcerative Colitis

Bicks, Kirsner and Palmer have reported some very interesting findings in regard to serum protein fractions this year[6, 7]. They have found 61 of 63 patients, presenting definite alterations in the electrophoretic patterns of their plasma proteins. The two exceptions were patients during the first week of a recurrence of their disease. They record two types of abnormality, one small group of patients showing hypoalbuminemia with increased A2 globulin and another larger group where these changes were present and accompanied by gross hypergammaglobulinemia. They interpret these findings as suggesting that increased antibody production does occur during the active phase of the disease. They also report that improvement with steroid therapy was most marked in the group characterized by the hypergammaglobulinemia. This was the larger group comprising forty-five patients. Steroids were found to be less effective clinically in the remaining patients, and they further suggest that when steroids were not effective in the patients exhibiting

hypergammaglobulinemia that, this was found to be a serious prognostic sign. These two short reports of work of undoubted excellence, certainly, give us good reason to hope for the development of more reliable criteria for the employment of steroid therapy in this disease in the near future.

Diseases Associated with The Liver

No particularly new discoveries were encountered by your gleaner. Papper et al[8] give an excellent review of an old problem, namely the occurrence of renal failure in cirrhosis of the liver. They have no new discovery to report.

Grant et al[9] bring some cheer to the cirrhotic in their report of a statistically significant diminution in the incidence of myocardial infarction in patients suffering from portal cirrhosis.

Manso, Friend and Wroblewski[10] imply a warning to those of us who are tempted to exhibit steroid therapy indiscriminately in liver disease. They find that "hydrocortisone may exert an adverse effect on the natural course of experimentally induced viral infection."

Enteric Fevers

K. C. Watson[11] reports an investigation on the nature of the relationship of relapses in typhoid fever to adequacy of treatment with Chloramphenicol. He found no qualitative difference between the nature of antibody produced in animals treated with Chloramphenicol and in non-antibiotic treated animals. Of necessity only certain aspects of these qualities were investigated. It is suggested that the higher relapse rates found in inadequately treated cases are not related to failure of antibody formation or qualitative alteration in the antibody but rather inadequate dosage. This article is most timely in view of our recent local flurry of interest in typhoid fever.

Wilson's Disease, that rare but fascinating disorder of copper metabolism mentioned here by virtue of its given name Hepatolenticular Degeneration by which it qualifies as a gastroenterological disease. No particularly new developments were encountered in this field during 1959, but Dr. J. M. Walshe[12] presents a brief but excellent and readable history in the November issue of the Annals of Internal Medicine. Space does not permit the abstraction of this editorial here, but in addition to its factual interest the history of this disease beginning with the recording of the pathological and clinical manifestations in 1912 followed by periodic reports upon investigations of its pathogenesis through the 1930's and the peculiar appropriateness of the development of British antilewisite during the early 1940's is in itself material to stir the imagination. Developments in this field since the release of information on BAL in 1946 have continued to be a fascinating "growing point" in the files of medical knowledge.

Pancreas

Allusion has already been made to the fact that Cystic Fibrosis of the Pancreas has now grown beyond the realm of the pediatrician and become of interest to the general practitioner and internist. It[13, 14] has now become important for us to include this condition in our consideration of differential diagnosis of chronic pulmonary disease in young adults. The large foul unformed steatorrheic stools, clinical appearance of malnutrition, combined with ravenous appetite completes the clinical picture. The much publicized "salt test" of the perspiration pretty well clinches the diagnosis and of course points up one of the other hazards of the disease—the exceptional susceptibility of these young adults to heat prostration due to excessive sodium chloride loss in hot weather.

Four criteria of diagnosis are quoted by di Sant'Agnese and Anderson[13] as follows:

1. Increase in concentration of electrolytes in sweat.
2. Absence of pancreatic enzymes on assay of aspirated duodenal secretions.
3. Chronic pulmonary involvement.
4. Family history of similar disorders. It is also remarked that untreated adrenal insufficiency is the only other known condition which will exhibit a similar electrolyte abnormality of sweat. The "sweat test" is therefore probably quite reliable.

It is hoped that these gleanings may have provided a new fact for some readers or have indicated a useful reference to others. It should also be mentioned here that the Surgical Clinics of North America published a symposium on recent advances in surgery of the gastro-intestinal tract in October, 1959. This was edited by Dr. Robert Turell. This pamphlet of 290 pages is an excellent volume with copious references and suggested supplementary reading.

References

1. Muggia, A., Spiro, H. M.: Childhood Duodenal Ulcer Gastroenterology, 37, 6, 715, 1959.
2. Scott, Norman M., Jr.: Experiences with the vigorous diagnostic approach to upper Gastro-intestinal haemorrhage. Annals of Internal Medicine, 51, 1, 89, 1959.
3. Bralow, S. P., Girsh, S.: Urticaria of the Gastric Mucosa with Massive Haemorrhage following Oral Penicillin Anaphylaxis. Annals of Internal Medicine, 51, 384, 1959.
4. Katz, Albert M.: Haemorrhagic Duodenitis in Myocardial Infarction. Annals of Internal Medicine, 51, 2, 212, 1959.
5. Varro et al: Experimental Phenylbutazone ulcer in dogs. Gastroenterology, 37, 4, 463, 1959.
6. Bicks, Kirsner and Palmer: Serum Proteins in Ulcerative Colitis.
   1. Electrophoretic Patterns in Active Disease. Gastroenterology, 37, 3, 356, 1959.
7. 2. The Effects of Therapy Correlated with Electrophoretic Patterns. Gastroenterology, 37, 3, 263, 1959.
8. Papper et al: Renal Failure in Laennec's Cirrhosis of the Liver. Annals of Internal Medicine, 51, 4, 759, 1959.
9. Grant et al: The Incidence of Myocardial Infarction in Portal Cirrhosis. Annals of Internal Medicine, 51, 4, 774, 1959.
10. Manso, Carlis, Friend, Charlotte and Wroblewski, Felix: The Influence of 17-Hydroxycorticosterone on Viral Hepatitis of Mice. J. Lab. and Clin. Med., 53, 5, 729, 1959.
11. Watson, K. C.: Effect of Chloramphenicol on Qualitative Aspects of Antibody Production Against Enterobacteriaceae. J. Lab. and Clin. Med., 53, 5, 743.
12. Editorial: Walshe, J. M. Annals of Internal Medicine, 51, 5, 1110, 1959.
13. Editorial: Annals of Internal Medicine, 50, 5, 1321, 1959.
14. Conference on Cystic Fibrosis of the Pancreas. Gastroenterology, 37, 4, 490, 1959.

The Management of Phlebitis Migrans*

S. G. Sheps, M.D., F.R.C.P. (C)†

A more descriptive but longer term for this condition is recurrent, idiopathic, superficial thrombophlebitis. This was first described in 1845. It usually occurs in men, in the third and fourth decade. It involves previously normal superficial veins. Pathological examination of excised segments of involved veins reveals no distinguishing features. It is commonly seen in the leg, but may be on the trunk or upper extremity. The phlebitis is of variable length but it is always associated with a firm, indurated cord surrounded by considerable local rubor. Occasionally discrete red nodules may be located next to these cords. It is fleeting, lasting one to three weeks. Occasionally there is a deposit of pigment to mark the site of the previous induration. Rarely this condition is associated with deep venous thrombosis and pulmonary infarction. The cause is unknown, but there have been attempts to relate this occurrence to allergy (a high percentage of patients have hay fever and eosinophilia), and to smoking. In a large series, however, cessation of the use of tobacco was not associated with any improvement[1]. The course is one of intermittent attacks over many years.

This type of phlebitis must be differentiated from local thrombophlebitis in a previously varicose vein. This differentiation is usually obvious. Secondly, it must be differentiated from thromboangiitis obliterans or Buerger's disease—this is usually associated with obliterative arterial lesions. In the absence of the latter it cannot be differentiated except perhaps by a response to cessation of the use of tobacco. Finally, it must be differentiated from phlebitis associated with occult carcinoma. This usually progresses in a rapid and relentless fashion. Moreover, this usually occurs in older people. One is justified in a thorough study to uncover cancer in any case in which two bonafide episodes of thrombophlebitis have occurred, regardless of the location, mildness or shortness of duration, provided there is no convincing explanation for the thrombophlebitis on some other basis[2]. A single bout of phlebitis should arouse suspicion if it is associated with suspicious systemic symptoms. Refractoriness of an episode of thrombophlebitis to adequate anticoagulant therapy should arouse suspicion of cryptic malignant disease. Myeloproliferative disorders (which include polycythemia vera, myelogenous leukemia, and thrombocytosis) may be grouped conveniently with occult cancer. These conditions can be associated with a hypercoagulable state, irrespective of the number of circulating platelets, and may first manifest themselves by recurrent phlebitis. There also may be an associated bleeding tendency, which makes the administration of anticoagulants hazardous.

A screening study to uncover occult malignancy includes a careful history and physical examination, with particular attention given to the thyroid, breasts, prostate and pelvis. A sigmoidoscopy would also be included. Roentgenographic examinations are made of the thorax, kidneys and gastrointestinal tract. Laboratory studies would include a routine urinalysis, complete blood count and sedimentation rate, blood sugar, lipase, amylase and leucine aminopeptidase. Estimations of cryoglobulins, cryofibrinogens, cold agglutinins, and a serum electrophoresis may uncover a dysproteinemia associated with a hypercoagulable state. Occasionally a markedly accelerated coagulation time is noted in these states. There are some who have a pessimistic attitude towards this type of study, claiming that all these cases are associated with disseminated carcinoma. However, it has been pointed out that the appearance of phlebitis may antedate the diagnosis of underlying malignant disease by some months[2]. Thus an earlier diagnosis may permit operation before the development of metastases.

The management of an acute episode of superficial phlebitis involves rest and elevation of the involved area, and the application of moist heat. Analgesics readily control the discomfort. The administration of anticoagulants is indicated when the involvement is extensive, or is at the sapheno-femoral junction or axillary vein. Deep venous thrombosis and pulmonary infarction are also indications for anticoagulant therapy. After the tenderness has subsided the patient may be allowed up with elastic support to the involved area. This support should be continued while the induration persists. When superficial thrombophlebitis is of a recurrent nature and the etiology is unknown, long term administration of anticoagulants is associated with a reduced incidence of thrombophlebitis and therefore a reduced morbidity. The duration of this anticoagulant therapy is unknown and should be individualized.

The significance of phlebitis migrans lies in its heralding of serious systemic disease. "All patients with phlebitis migrans should be re-examined periodically for evidence of occlusive arterial lesions, carcinoma, or blood dyscrasia even though these were not detectable at the time of the first examination."[1]

References

1. Allen, E. V., Barker, N. W., Hines, E. A.: Peripheral Vascular Diseases. W. B. Saunders Co., Philadelphia. Second Edition, 1955.
2. Wooling, K. R., Shick, R. M.: Proceedings of the Staff Meetings of the Mayo Clinic. Vol. 31: 227-233, April 18 1956.

* Based on contributions to a panel discussion of "Phlebitis and Thrombosis — Practical Management," at the 1959 Annual Meeting of the Manitoba Medical Association.

† From the Department of Medicine and Cardiology, Manitoba Clinic, Winnipeg, Manitoba.

Case Report

Bilateral Ovarian Apoplexy in Lupus Erythematosus

Murray Campbell, M.D.

In an extensive report on abdominal emergencies in systemic lupus erythematosus fourteen cases are reviewed[1]. In ten with lupus peritonitis the abdominal symptoms were attributable to vascular lesions in the wall of the bowel and its appendages or similar disease in the retroperitoneal, peripancreatic or periadrenal connective tissues; in some the lesions were localized, in others widespread. Every patient complained of abdominal pain. Nausea, vomiting, fever and tachycardia were common. Tenderness was usual and rebound tenderness frequent. In three patients a diagnosis of acute appendicitis was made because of rebound tenderness in the right lower quadrant. Along with acute appendicitis, paralytic ileus, parametritis, obstruction of the small bowel and perforating peptic ulcer, many other diagnoses were made. The remaining four were diagnosed as acute pancreatitis, and indeed three of these had lesions in the pancreas. One patient had both polyarteritis and lupus nephritis proven at autopsy. In another report[2] chronic or subacute gastro-intestinal symptoms constituted the chief complaint in 5 out of 56 cases, and these were due to vascular involvement of the bowel with infarction or hemorrhage. In a monograph on the whole subject of systemic lupus erythematosus[3] 38 out of 138 cases had moderate or severe gastro-intestinal complaints and 4 had hemorrhage from the bowel, one of whom died of anemia from bloody diarrhoea.

The present case, a sixteen year old white girl had severe abdominal pain of sudden onset for twelve hours prior to admission to hospital September 4th, 1958. The pain of an intense colicky character began in the left lower quadrant, but when seen it was in the lower right side. It did not radiate. The tenderness was maximal just above the pubis and to the right. There was no rigidity and bowel sounds were present. Rectal examination suggested a fluctuant mass in the right cul-de-sac. The appendix had been removed. The patient had just completed a protracted period and had been very tired for several days. She stated she had rheumatoid arthritis for two or three years and this was supported by the presence of two spindle-shaped interphalangeal joints. Two weeks before admission "cysts" had been removed from the left knee and the right wrist at another hospital and while the scars substantiated this, there were no gross abnormalities seen. A rash on the nose and cheeks had been present for two months. While not in shock the patient looked very pale and very sick. The heart and lungs were normal, and the blood pressure 110/60. The hemoglobin was 41%, the total leucocyte count, 6,800 with a normal differential and normal bleeding, clotting and prothrombin times. The sedimentation rate was 100 mm. and the urine showed a few hyaline and granular casts.

The most likely diagnoses were considered to be hematosalpinx, and ectopic pregnancy, and laparotomy was decided upon. At operation the abdomen was full of blood, some 2,000 cc.s being removed. The chief source of bleeding was the right ovarian artery and there was also some oozing from the left ovary. The right ovary was removed, the left sutured and no other bleeding points were observed. The other organs appeared normal. The surgeon (Dr. Kenneth Trueman), suggested the term ovarian apoplexy. The patient's condition was critical, but she responded well to transfusions. She developed moderate fever and a degree of atelectasis at the base of the left lung. This cleared with antibiotics and she was able to be out of bed briefly six days later. Because of the erythematous rash on the face and the unusual operative findings further investigation was immediately carried out; a high gamma globulin and one doubtfully positive L.E. cell test were found. The microscopic examination of the ovary removed at operation was not helpful. There was edema, and hemorrhage with round cells predominating-polymorphs and eosinophils were rare, and no conclusive diagnosis was made.

On the eighth day bloody discharge started coming from the wound, and two days later she had a severe nose bleed. The bleeding and clotting times were still normal and remained so until death, as did the platelet count. However, the prothrombin time was now 62% of normal and shortly before death it had dropped to 10% of normal. The patient was placed on 40 mgm. of prednisone daily with no change in her symptoms. She was then switched to medrol 32 mgm. daily and within 24 hours the oozing had stopped, the temperature which had been elevated fell to normal and for two days the patient was ambulant. Unfortunately and unexpectedly the symptoms recurred, and prednisone 60 mgm. daily had little effect. Another change was made to large doses of solucortef intravenously with slight improvement. Throughout the stay in hospital the institution of intravenous therapy and cut-downs was extremely difficult and the assistance of the anesthetists in this respect was invaluable. On October 4th, one month after admission, a fluctuant mass was again found in the pouch of Douglas, and a considerable amount of blood was evacuated through a colpotomy. Melena had been present for some days and at this stage the patient began to pass bright blood by rectum and vomit blood as well. Death occurred four days later with evidence of paralytic ileus. Permission for autopsy could not be obtained.

A dermatologist, two hematologists and an otolaryngologist had seen the patient. It was

agreed that systemic lupus erythematosus was the most likely diagnosis and that the patient had an unusual circulating anticoagulant or a fibrinolysin which caused the bleeding. Such an anticoagulant has been described[3] in systemic lupus and was considered to be associated with an abnormal globulin, but unlike the present case it was in conjunction with prolonged clotting times. In some respects the diagnosis of periarteritis nodosa would have been more compatible with the symptoms, but the blood pressure was never above normal limits.

References

1. Pollak, E. V. et al: Systemic Lupus Erythematosus Simulating Acute Surgical Conditions of the Abdomen. New Eng. Jour. Med., 259: 6, p. 258-265, 1958.
2. Dubois, E. L.: Effect of L.E. Cell Test on Clinical Picture of Systemic Lupus Erythematosus. Ann. Int. Med., 38: 1265-94, 1953.
3. Harvey, A. McGehee: Systemic Lupus Eruthematosus. Medicine, 33: 291-423, 1954.

Surgery

Surgical Aspects of Pulmonary Emphysema

J. M. Kagan, M.D., F.R.C.S.(Can.)

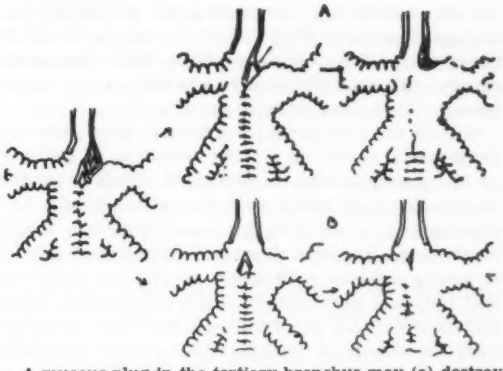
One of the commonest causes of chronic pulmonary failure is pulmonary emphysema. The term emphysema of the lung indicates over-inflation of the pulmonary tissue; the over-inflation involves the alveoli and distal parts of the bronchioles. The lungs may be involved in different regions and to varying extent; the process may be diffuse and bilateral or unilateral involving the lobar or segmental regions. This article will discuss emphysema under three forms; generalized bilateral vesicular emphysema, lobar emphysema and emphysematous bullae.

Pathogenesis

Over-inflation of the lungs occurs whenever there is a partial obstruction in the air passages; the obstruction is such that during inhalation air enters easily into the lung but during exhalation the air is stopped or is expelled with difficulty and becomes "trapped" in the distal regions of the lungs. The trapped air produces over-distention or emphysema of the involved portions of the lungs. The extent and the localization of the emphysematous process depends on the region and the level of the obstruction.

In diffuse vesicular bilateral emphysema the obstruction is found in the bronchioles. To understand the pathogenesis of diffuse emphysema the anatomy of the terminal bronchioles must be kept in mind. The bronchial tree after dividing dichotomously many times ends in the terminal bronchi which lead to the respiratory bronchioles. The respiratory bronchioles undergo three orders of division before they terminate in alveolar ducts, which, in turn, open into alveolar sacs. The walls of the respiratory bronchioles are composed of an inter-lacing mesh of musculo-elastic tissue forming the mouths of alveoli, whereas the alveolar duct walls are composed of an open mesh which terminates in alveolar sacs.

There are inter-alveolar pores (Cohn), by which adjacent alveoli and adjacent respiratory bronchioles communicate.



A mucous plug in the tertiary bronchus may (a) destroy the bronchus thus obliging the airflow into the distal alveolus to take a circuitous route through the inter-alveolar pores of cohn with enlargement of the pores and breakdown of interalveolar wall or (b) expulsion of plug will result in less interalveolar-wall breakdown.

Patency of the airways depends on the efficiency of the homeostatic mechanisms of the host; that is, there is a balance between the ciliary action, cough and collateral ventilation on the one hand, noxious agents such as viral and bacterial infections and chemical irritants on the other. Temporary occlusion of the small bronchioles by mucous is an every day event. This occlusion usually occurs at the level of the primary respiratory bronchiole. Material aspirated beyond the primary respiratory bronchiole is usually phagocytosed and aeration of the passages distal to the occlusion is maintained by collateral ventilation through the inter-alveolar pores. As long as aeration is maintained beyond the occluded bronchiole cough will expel the bronchial plug.[11] Cigarette smoking is the commonest cause of terminal bronchiolitis, for the irritating smoke is able to penetrate deeply into the terminal air passages.[12, 13]

Should the bronchiolitis be too severe for the homeostatic mechanisms to disengage the bronchiolar plug, a sequence of events occurs which terminates in vesicular emphysema. If the bronchiolar plug is permanent, "air trapping" occurs in the air passages distal to the occlusion; the air enters the alveoli by collateral ventilation during inspiration through the alveolar pores. Since the passage

is long and tortuous and since passages and pores diminish in size during expiration air becomes trapped beyond the point of occlusion. The continuous distention of the passages beyond the occlusion causes a disruption of the walls of the alveoli by dilatation of the inter-alveolar pores and thinning of the alveolar walls.

As a consequence of the disruption of the alveolar walls and respiratory passages beyond the occluded bronchiole there occurs the following: an increased freedom of collateral ventilation in the affected area so that air trapping is reduced; in the more advanced lesions involving a considerable segment of pulmonary tissues, the remaining bronchioles open into a "common pool" of dilated and disrupted alveoli. This "pool" is usually found in the centre of the secondary lobule. Thus disruption occurs until equilibrium is reached at a much lower pressure than that existing initially.[11]

Should the obstructing mechanism producing the emphysema occur in a lobar bronchus, a regional or lobar emphysema is produced. Such a partial obstruction may occur with the presence of a foreign body or a tumor in the lower bronchus. Lobar emphysema has been described in newborn infants or young children with severe respiratory difficulty and cyanosis unrelated to acute infection or other known event. An abnormality of the lobar bronchus such as abnormal collapsibility of the bronchial walls is thought to be the important factor in production of this syndrome.[5] A partial stenosis of the left lower lobe bronchus producing left lower lobe emphysema has been described to occur in patients with chronic fibrotic tuberculosis of the left upper lobe. The contraction of the fibrotic left upper lobe produces a kinking of the left main stem bronchus under the arch of the aorta producing a functional stenosis to the lower lobe bronchus[6, 12].

Due to secondary pathological processes in the lungs, such as loss of elasticity due to scarring and inflammation or occlusion of the inter-alveolar pores by granulomatous lesions, a focal lesion of emphysema or the common pool may be restricted to a certain region to form an emphysematous bulla. When this emphysematous focus is localized entirely within the pulmonary parenchyma it is called a bulla, if it is found under the visceral pleura it is called a bleb.

Management:

The surgeon is variously concerned with emphysema depending on its extent and localization.

Diffuse vesicular or hypertrophic or obstructive emphysema:

In order to avoid operative and post-operative complications it is important for the surgeon to recognize the presence of pulmonary failure due to generalized emphysema in patients who are candidates for surgery, both thoracic and abdominal. Diffuse emphysema is diagnosed from the history, physical examination and laboratory tests. The patient has a history which begins with slight limitation of activity as a result of exertional dyspnea. Chronic cough with marked exacerbation of dyspnea associated with acute respiratory infections is followed by progressive dyspnea which finally becomes incapacitating. An acute respiratory infection may be followed by intolerable dyspnea, severe cyanosis and evidence of right heart failure.[4] On Physical Examination a barrel chest deformity and a slight cyanosis due to decreased oxygen saturation of the blood may be noted. Distension of the neck veins, hepatomegaly, and peripheral edema will indicate the presence of right heart failure.[4] X-ray examination of the chest will reveal low, flat diaphragms with increased translucency of the lower lobes, increased vascular markings at the hila and reticular formation in the peripherap lung fields[8]. Fluoroscopy will reveal diminished motion of the diaphragm. Occasionally emphysematous bullae difficult to visualize on the Roentgenogram may produce a relatively normal looking chest plate in a patient who is dying of pulmonary emphysema. Lung volume studies show a change in the lung compartments: the residual volume is increased, the vital capacity decreased, and the total capacity normal or increased. There is an increase in the resistance of the flow of air probably related to unequal flow rates in various bronchioles, turbulence of air associated with bullae, or actual narrowing of the bronchioles. Bronchial infection greatly increases airway resistance. A study of the blood gases shows a decrease in oxygen saturation of arterial blood and an increase in carbon dioxide tension. This is due to interference with even distribution of air, as well as uneven perfusion of the alveoli. Occasionally the blood alveolar gas exchange may be so disturbed as to cause an abrupt rise in carbon dioxide tension and a fall in pH or respiratory acidosis. This may give rise to headache, somnolence, semicoma and peripheral muscular twitchings. A mistaken diagnosis of brain tumor has been made in cases of respiratory acidosis due to pulmonary emphysema.[1] In the emphysematous patients normal response of the respiratory centre to carbon dioxide tension is lost; hypoxia through the medium of the chemoreceptors of the carotid and aortic bodies serve as an effective stimulus to ventilation. Reduction of hypoxia by the administration of oxygen may produce an oxygen apnea and a subsequent carbon dioxide narcosis. Therefore, oxygen must always be administered by an artificial respirator machine, or by an intermittent positive pressure machine to an emphysematous patient.

Pre-operative management of patient with diffuse emphysema:

The management of the patient with pulmonary failure resembles the management of patients with

congestive heart failure in the following sense; it is difficult to alter the basic pathology but often possible to make the patient a tolerable operative risk. Since the emphysematous patient has best pulmonary efficiency during quiet breathing it is important to dispel anxiety[2]. A period of abstinence from cigarette smoking is mandatory. Some authors believe cigarette smoking to be the pathogenic agent of emphysema[3]. Treatment of chronic bronchitis with appropriate antibiotics might be instituted.

Oxygen administered by intermittent positive pressure breathing, using the Bennett IPPB/I machine with the automatic flow sensitive or pressure sensitive respiratory valve (Bennett TV-2P valve) combined with a broncho-dilator such as Vaponephrin or Isuprel for thirty minutes two or three times a day for four or five days pre-operatively, greatly improves the patient's condition. Pancreatic dornase, a desoxryleonuclease of pancreatic origin, and a suitable antibiotic can be combined in the aerosol treatment in patients with thick tenacious sputum.[14]

Post-operative care of patient with diffuse emphysema:

This should include the following necessary measures: avoidance of depressing narcotics and sedatives; oxygen therapy to be administered with the intermittent positive pressure breathing machine, which may be combined with the aerosol broncho-dilators. The intermittent positive pressure breathing may be combined with a negative expiratory phase by the use of the "Cof-flator" or the "Exsufflator" to help expel the bronchial secretions in patients too old and too weak to cough properly.[3] The head down position improves bronchial drainage and diaphragmatic motion. Where the emphysema is combined with severe bronchitis and copious bronchial secretions a Tracheotomy may be considered to insure an airway free of secretions and to diminish dead airspace.[7]

Surgical Treatment of Localized Emphysema

Surgical removal of localized emphysematous segments may be indicated to correct mechanical interference with ventilation produced by the distended and non-functional portions of the lung. Emphysematous lobes and giant emphysematous bullae should be removed surgically. As mentioned above, extreme lobar emphysema has been found in infants and small children. It is a dramatic condition in which a single lobe which is grossly normal becomes tremendously dilated occupying the entire hemi-thorax, compressing the adjacent lobes and pushing the mediastinum to the opposite side. Resection of this emphysematous lobe leads to restoration of a relatively normal respiratory status.[5] Emphysematous bullae caused by the breaking down of inter-alveolar septa within the lung parenchyma occasionally may increase to such a size as to compress adjacent lung with interference of function. Giant cysts pneumatoceles, have occasionally been found in infants and children. Conservative resection of the bullae permits the collapsed and compressed portions of the lung to re-inflate. Here the surgery consists in opening the major cyst or cysts with removal of the peripheral portions. The inner wall will be seen to consist of compressed and attenuated trabeculated lung with multiple pin-point fistulae; these are closed with purse string sutures of fine silk, and the lung substance approximated progressively by interrupted mattress sutures to obliterate the cavity.[9] Occasionally a sub-plural bleb may burst and produce a spontaneous pneumo-thorax. If the pneumo-thorax is large, over 20% collapse, it may be reduced by repeated thoracentesis or inter-costal water sealed drainage. Should the pneumothorax become recurrent, a pleurodesis may be carried out.

Summary

When a patient with diffuse vesicular emphysema is a candidate for major surgery the surgeon must know and adopt essential pre-, and post-operative measures to avoid pulmonary complications.

A patient with lobar emphysema, emphysematous blebs and bullae, may be successfully treated surgically.

References

1. Neurologic Manifestations of Chronic Pulmonary Insufficiency: Frank K. Husten, Miriam W. Carmichael, Raymond O. Adams et al. New England Journal of Medicine, 257: 579 (Sept. 26) 1957.
2. Pulmonary Emphysema: A. L. Barack, H. A. Bickerman. Baltimore: William & Wilkins Co., 1956.
3. Physical Methods Simulating Mechanism of the Human Cough: Alvan L. Barack et al. Journal of Applied Physiology, 5: 85, 1952-53.
4. Pulmonary Failure: Richard V. Ebert. Disease-a-Month, p. 11, October, 1958, Year Book Publishers Inc., Chicago.
5. Lobar Emphysema: Harry W. Fischer, Joseph Cucido and Chester Lynxwiler. J.A.M.A., 166: 340 (Jan. 25) 1958.
6. The Patterns and Dynamics of Trachiobronchial Deformity in Pulmonary Tuberculosis: G. A. P. Hurley, D. Todosijczuk. Journal of Thoracic Surgery, 37: 166 (Feb) 1959.
7. Tracheotomy as an Adjuvant to Abdominal Surgery: J. M. Kagan. Manitoba Medical Review, 37: 230, 1957.
8. Radiological Diagnosis of Emphysema: J. N. S. Knott, Ronald V. Christie. Lancet, 1: 881, 1951.
9. Thoracic Surgery and Related Pathology: Lindskog & Liebrow, p. 345, 19. Appleton-Century-Crofts Inc., New York, 1953.
10. Chronic Obstructive Pulmonary Emphysema: A Disease of Smokers: Francis C. Towell et al. Annals of Internal Medicine, 45: 260, 1956.
11. Pathogenesis of Pulmonary Emphysema: K. H. McLean. American Journal of Medicine, 25: 62 (July) 1958.
12. Functional Stenosis Due to Bronchial Angulation: C. H. Nicholson et el. Journal of Thoracic & C. U. Surgery, 38: 90 (July) 1959.
13. Chronic Bronchitis: A neglected disease entity: R. W. Phillips et al. Diseases of Chest, 26: 520, 1954.
14. Intermittent Positive Pressure Breathing: Its use in the inspiratory phase of respiration. M. S. Segal et al. New England Journal of Medicine, 250: 225, 1954.
15. Carbon Dioxide Intoxication: The clinical syndrome, its etiology and managements with particular reference to the use of mechanical respirators: H. U. Silker, J. B Heckman. Medicine, 35: 389, 1956.

Lymphedema Praecox

Morris H. Broder, M.D.

Lymphedema is an abnormal accumulation of lymph in the tissue spaces. Despite the simplicity of this definition, general information about lymphedema has been somewhat confused. Furthermore, the term has been loosely applied to a variety of conditions which produce a large extremity. For these reasons a case report and a brief review of the subject of lymphedema with particular reference to lymphedema praecox appears indicated.

Case History

Mr. H. A. was admitted on March 19th, 1958, to the Misericordia Hospital for the investigation and treatment of lymphedema of the left lower limb. He gave the following history:

In November, 1951, when 12 years old, the patient was in a fight. While being held down, a 14 year old girl jumped on his left lower limb several times, bruising the front and back of the left thigh. Two weeks later the patient went to bed because of the bruises and the mother noticed that the entire left lower limb was swollen from the ankle to the hip. This swelling remained, although it would partially subside during the night.

Examination in 1953 showed pitting edema of the left lower limb extending proximally to the hip. There was no increased heat, and leg length was equal on both sides. The left thigh was 5 inches greater in circumference than the right. The patient was advised to wear an elastic support and was also instructed as to the general care of this enlarged extremity. He was again seen in 1955, and it was then noted that the left thigh was now 7½ inches greater in circumference than the right, and the left calf was 5½ inches greater in circumference. When seen in March, 1958, the patient complained bitterly that the leg tired easily and that he could hardly drag it around. He also stated that the leg and thigh were steadily increasing despite the elastic support (rubber bandaging). Examination at this time showed a further increase in size; the left thigh and calf now were 10½ inches and 8½ inches greater in circumference than the right. Femoral pulsations were equal. However, the popliteal posterior tibial pulses were not palpable, probably because of the difficulty in palpating them through the thick lymphedematous tissue. Leg length was again noted to be equal and there was no increased heat over the extremity. There was no audible or palpable bruit.

General physical examination was essentially negative. A complete blood count and sedimentation rate, and a urinalysis was normal. X-rays of the chest and long bones were also normal. Because of the history of trauma, femoral arteriograms and venograms were obtained which showed an essentially normal arterial and venous system.

It was decided that surgical intervention was now indicated because of the steady progression and the fact that the edema was now solid (it did not pit on pressure). Furthermore, it was quite obvious that the patient was willing to go to any measures to have this limb reduced in size. Discussions with the boy's parents also revealed that he was becoming a behavior and social problem because of his self-consciousness regarding the limb. In Figure 1, the pre-operative appearance of the limb is shown. In March, April and May of

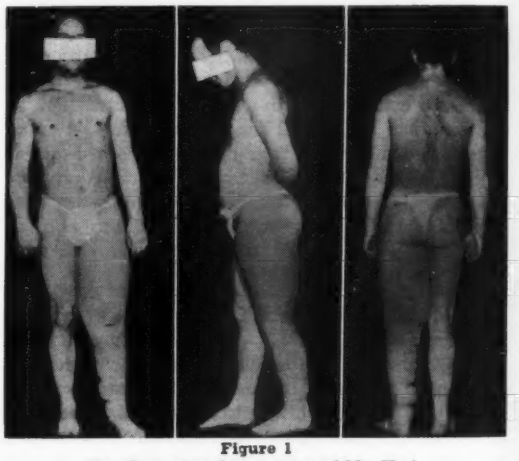


Figure 1
Pre-Operative Appearance of Mr. H. A.

1958, a three stage procedure was performed in which the subcutaneous tissues, fat and deep fascia were excised and the skin replaced as full thickness skin graft. The excision extended from the metatarsal-phalangeal joints to just above the knee. The technique followed was essentially that described by Pratt. In Figure 2 — the post-operative appearance taken six months following the last procedure is shown.

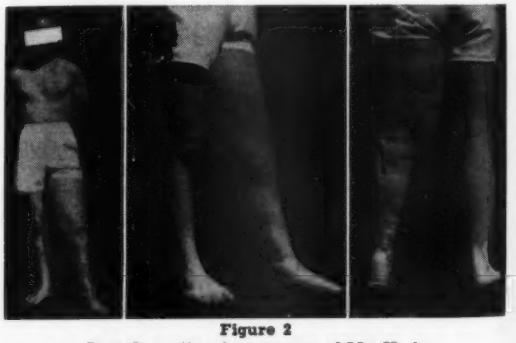


Figure 2
Post-Operative Appearance of Mr. H. A.

Histological examination of representative sections showed enlarged lymphatic vessels, fibrous connective tissue, collagenous in some areas and an edematous and myxoid appearance in others.

Perivascular inflammation with perivascular infiltration with chronic inflammatory cells was also noted.

Examination in November, 1958, showed that the left calf (operated side) was now 1 inch smaller than the right, and the circumference at both ankles was equal. The patient expressed much gratitude and felt that it was well worthwhile despite the long hospital stay. He is to return for further staged procedures on the left thigh.

Discussion

Although this paper is primarily concerned with lymphedema praecox, one cannot help but introduce a few pertinent remarks which apply to lymphedema in general.

Our knowledge of the physiology of lymphedema leaves much to be desired. The lymphatic system complements the return of water, electrolytes and waste products to the blood stream. Under ordinary circumstances, lymph is the protein free filtrate of the blood plasma, the crystalloids being fairly permeable to the capillary membrane in both directions. The formation and removal of lymph in its simplest form is illustrated in Figure 3. Here we

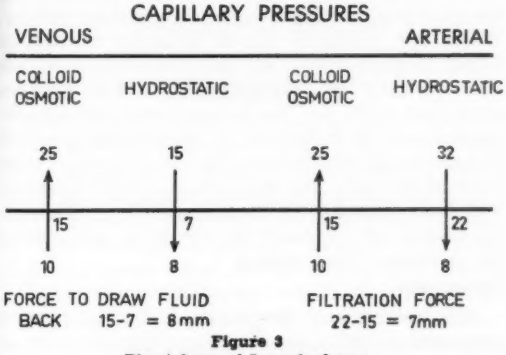


Figure 3
Physiology of Lymphedema.

see that the hydrostatic and colloid osmotic pressures at the arterial and venous end of the capillaries are in a state of delicate balance. The net result is that most of the lymph that is filtrated at the arterial end of the capillary is absorbed at the venous end. The lymphatic fluid absorbed by the lymphatic capillaries is not depicted in Figure 3. Thus, lymph can accumulate in tissue spaces either by excessive formation or decreased removal. The increase in formation of lymph may be due to a variety of causes, such as damage to the capillary wall (anoxia, inflammation, trauma, burns, cold), low osmotic pressure (plasma proteins reduced due to disease of the kidney, liver, starvation, etc.). The central causes of lymphedema, such as diseases of the heart, kidneys and liver act basically through the factors of increased hydrostatic pressure at the venous end of the capillary, and the reduced osmotic pressure of the plasma proteins. The local causes of lymphedema are essentially due to venous obstruction, lymphatic obstruction or both. Unfortunately, there is no uniformity of opinion as to which of these factors is the most important. According to the studies of Drinker, the veins can compensate for lymphatic obstruction, but the lymphatic system cannot compensate for venous obstruction. Experimentally complete excision of the iliac lymph nodes does not produce edema. Conversely one does not always get lymphedema when the venous return of an extremity is compromised by ligation of the main venous channels. Some investigators have experimentally excised across an entire limb and sutured the skin, leaving only the artery and vein intact, to find that lymphedema is only temporary. On the other hand, repeated injection of silica into the lymphatics to produce lymphatic obstruction will produce lymphedema. Perhaps regeneration or the opening of new lymphatic pathways in lymphatic obstruction, or the opening of new venous collaterals in venous obstruction will obviate lymphedema from occurring.

Most surgeons who are called upon to treat lymphedema well realize the difficulties encountered because of the tendency to progression and irreversibility. An attempt is made to illustrate the pathological physiology of lymphedema in the cycle illustrated in Figure 4. Obstruction of the venous or lymphatic systems, due to inflammatory or noninflammatory causes will produce lymphedema or exudation of lymph into the tissue spaces. The exudate has an increased protein content which stimulates fibrosis; as the fibroblasts proliferate rapidly in this excellent culture medium. The resulting fibrosis still further aggravates the stasis. The fluid medium with its high protein content affords a good pablum for bacteria, so that recurring attacks of acute inflammation are frequent, leading to further stasis. Thus a vicious cycle may be established as indicated by the arrows in Figure 4. The edema is first pitting in character,

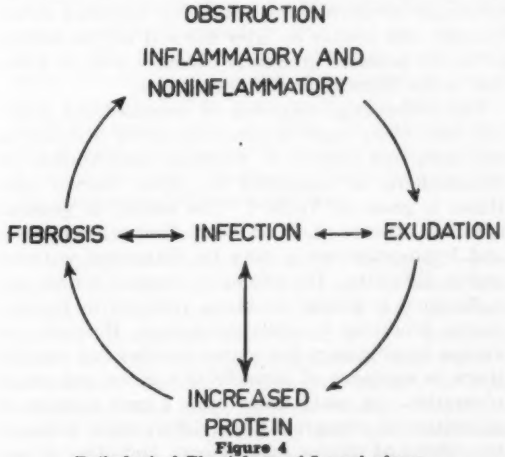


Figure 4
Pathological Physiology of Lymphedema.

but as cellulitis and fibrosis occur, the edema progresses to a solid phase. When one incises such a limb, superficially there is a free flow of lymph, deeper still there is an admixture of lymph and flecks of fibrin, and finally superficial to the deep fascia there is a hypertrophy of the fibrous connective tissue for a variable depth.

Lymphedema praecox is a relatively rare condition. According to Allen, Baker and Hines a diagnosis of lymphedema praecox was made in 93 of 300 cases seen at the Mayo Clinic. The unduly high incidence of lymphedema praecox in this series is probably a result of a selected group of patients. Probably the commonest cause of lymphedema one sees in practice is due to venous insufficiency or venous thrombosis with its sequelae.

The etiology of lymphedema praecox is obscure. The dilated lymphatic channels, and the fact that the edema does not occur usually until puberty, appear to indicate that the basis is one of lymphatic obstruction. Congenital underdevelopment of the lymphatics with their inability to keep pace with the growing limb may also be a factor. Gravity has also been implicated, as the disease invariably occurs in the lower limbs.

The clinical picture of lymphedema praecox is quite variable. Approximately 90% of cases occur in females, and approximately 70% affect only one leg and occur at the time of puberty. The onset is usually gradual commencing about the region of the ankle and progressing proximally. However, as noted in the case history the onset may be sudden and may progress very rapidly. Occasionally the lymphedema extends proximally to the region of the knee and appears to remain stationary at this point. The complications of lymphangitis cellulitis and ulceration are infrequent. As with other causes of lymphedema rest in bed and elevation of the extremity produce temporary disappearance of the edema. However, the swelling, whatever its limitations, gradually becomes more marked, and sooner or later the soft edema which pitted on pressure gradually becomes solid in form due to the fibrous tissue hypertrophy.

The differential diagnosis of lymphedema praecox from other types of edema is rarely difficult in the advanced stages. A working classification of lymphedema as suggested by Allen, Barker and Hines is given in Table 1. The edema of general systemic disease such as heart failure, nephrosis and hypo-proteinemia may be diagnosed without undue difficulty. The edema of chronic venous insufficiency is similar in many respects to lymphedema produced by other conditions. However, in venous insufficiency the edema is softer and usually there is evidence of superficial varices and stasis ulceration. In early cases only a past history of phlebitis and venography will differentiate between the edema of venous insufficiency and other types.

Table 1
Classification: Lymphedema

Noninflammatory

Primary
- Lymphedema Praecox
- Congenital Lymphedema
  - Hereditary or Familial (Milroys Disease)
  - Simple

Secondary
- Malignant Occlusion
- Surgical Removal of Lymph Nodes
- Pressure
- Roentgen and Radium Therapy

Inflammatory

Primary (Single or Recurrent Acute and Chronic)

Secondary (Single or Recurrent Acute and Chronic)
- Venous Insufficiency
- Trichophytosis
- Systemic Disease
- Filariasis
- Local Tissue Injury or Inflammation

Generally speaking a complete history and a thorough general physical and local examination is usually all that is required in a differential diagnosis of the local causes of lymphedema.

When the swelling of an extremity is localized, one must consider other conditions such as sarcomas, lipomas and bone tumors as possible causes. X-ray studies of the area may be indicated. Angioneurotic edema is characterized by recurrent attacks of lymphedema, accompanied by itchiness, minimal pain, sudden onset and an allergic history. When the extremity shows presence of dilated veins, increased heat and increase in length, one must suspect an arteriovenous fistula. Oxygen saturation studies of the venous blood and arteriography may be indicated. In lipodystrophy, which commonly attacks women and may also produce painless swelling, the condition is usually bilateral, and is associated with obesity elsewhere. There is no history of recurrent lymphangitis and pitting on pressure is less evident.

Treatment

The treatment of lymphedema praecox in most instances is still a most taxing problem. Medical treatment, to be effective, must be instituted when the edema first becomes evident, and even despite a proper medical regime carried out on a co-operative patient, the edema may still progress and reach the stage of irreversibility. Nevertheless, the longer the lymphedema is uncontrolled, the more fibrosis develops, and the less efficient medical treatment becomes. Essentially the problem is one of preventing stasis. At first the extremity is elevated until as much lymph as possible has been removed. Following this the patient wears an elastic support, preferably of a rubber roller bandage type, which will not stretch and give way in time. The patient should become adept at bandaging his leg efficiently. Lymphangitis and cellulitis to which the edematous limb is prone, should be treated vigorously and as early as possible. Unfortunately, there is no proven way of preventing these attacks other than control of the lymphedema. The protein rich lymphedematous fluid serves as a perfect pablum for bacterial growth.

Surgical measures for the treatment of lymphedema should be considered when the edema is irreversible and has reached a solid stage. A review of the surgical measures described to combat and correct lymphedema are not in the realm of this paper. The operations devised and performed to promote new lymphatic pathways, such as the implantation of silk threads in the subcutaneous fascia, abdominal flaps, etc., have all been uniformly unsuccessful. The various techniques such as described by Poth, Macey, Pratt, etc., all have in common the removal of the subcutaneous tissue and deep fascia which is the cistern in which the edema fluid collects. Because the procedure is of some magnitude, staging is often necessary, as in the described case history, to avoid shock. Women are often satisfied if the operation is performed from the toes to the knee (to the level of the skirt line). The heel, and sole of the foot do not suffer from edema in these cases, and dissection in these regions is unnecessary. The cosmetic result of surgery, although not entirely satisfactory, will produce an acceptable looking extremity in most cases. It should be stressed, that post-operatively the patient must continue to wear an elastic support. Women in particular will object to this. However, surgery in conjunction with an elastic support post-operatively will produce a significant improvement both from the cosmetic and functional aspects, in the grossly enlarged and irreversible lymphedematous extremity.

Summary

1. A case of lymphedema praecox has been presented.

2. The physiology and pathological physiology of the lymphedema has been briefly reviewed.

3. The etiology, differential diagnosis and treatment of lymphedema praecox has been briefly presented.

Urology

Perinephric Abscess

Melville J. Swartz, M.D., C.M.
Department of Urology, Mall Medical Group, Winnipeg, Manitoba

Perinephric abscess is of great concern to any and all interested in diseases of the kidney. Although the majority of cases are now recognized by certain well-known signs obtained by making the present day urological investigations, there still remains a group of cases in which the diagnosis is difficult. It is this group which produces the high mortality rate of the condition and which should act as a stimulus to effect its early diagnosis.

The average length of time taken to diagnose the condition is 35 - 40 days. The actual time in individual cases varies from three weeks to three years. In a large series of cases of perinephric abscesses reported upon, there is a fairly constant 30 - 35% mortality rate. Autopsies in these cases revealed the unsuspected lesion either as a direct cause or as a fatal complication of the disease. Some of these fatal cases have been treated on a urological service and one reported case came to postmortem after having two urological examinations.

Despite numerous papers on the value of the various signs and the most diligent efforts by urologist, surgeon, physician and radiologist to bring the findings into sharp focus, none can be said to be absolutely and specifically pathognomonic of perinephric abscess.

While most cases of perinephric abscess may be diagnosed with ease, there are cases in which this may be quite impossible, short of exploratory incision.

Perinephric abscess has been diagnosed and treated as influenza, typhoid fever, endocarditis, tuberculosis, meningitis, bronchitis, ecchinococcus cyst, intercostal neuralgia, cholecystitis, hypernephroma, tuberculosis of the spine, surgical knee, sacroiliac disease, psoas abscess, hip joint disease, malaria, gastroenteritis, diabetes, chronic myocarditis, prostatic abscess, empyema, pyonephrosis, acute pancreatitis, septicaemia, amoebic liver, septic hip, appendiceal abscess, neoplasm of the ilium, perforated duodenal ulcer, abscess of the groin, carcinoma of the colon, pneumonia, neuritis, intestinal obstruction, salpingitis, necrosis of the ribs, kidney abscess, carcinoma of the kidney, lipoma, lymphoma, retroperitoneal abscess, subphrenic abscess, osteomyelitis, ruptured kidney, nephrolithiasis, hernia of Petit triangle, tropical fever, pyelitis, rheumatism, myositis, etc., etc. The above fifty diagnoses were made in cases which were eventually diagnosed as perinephric abscess and were taken from a series of eight reports presented between 1950 and 1956. This should serve to emphasize the difficulty encountered in making an early diagnosis, which is so important in reducing the known high mortality rate.

Diagnosis

Perinephric abscess usually develops rather slowly and does not manifest itself for some time, usually being discovered after it has produced general symptoms characterized by persistent fever, obscure abdominal symptoms and signs of walled-off infection. A careful history is most important. The onset of fever usually coming on with a chill accompanied by pain and tenderness in the loin or upper abdomen; a high leucocyte

count (10,000 - 40,000); signs of psoas spasm, evidenced by changes in posture, flexion of the thigh; anorexia, nausea and vomiting, loss of weight, occurring a few days to a few weeks after some skin infection, tonsillitis, prostatitis, in which there is very little to be found on urinalysis, should make one suspicious of perinephric involvement.

Of all the signs and symptoms, fever is the most constant and reliable in arriving at a diagnosis. It usually comes on with a chill, is remittent in type, and, because of its persistent elevation, is often confused with other suppurative conditions. Pain is variable, usually constant, dull, non-radiating, located in the flank or upper abdomen. In some cases, the pain may radiate to the abdomen, umbilicus, scrotum or shoulder.

Fullness of the loin or abdomen is an important late diagnostic sign. When this occurs it is usually indicative of frank pus and does not move with respiration or changes in position. This fullness can sometimes be detected very easily by taking measurements from the spinous processes to the umbilicus and on both sides. The urine shows very little as a rule — possibly a trace of albumen and an occasional leucocyte or erythrocyte. Needling for purposes of diagnosis can be most helpful. This procedure is mentioned for purposes of discussion amongst the readers, as many consider it a dangerous procedure. This, of course is a matter of opinion, and like trans-abdominal aortography, those who find it a valuable asset should use it and those who consider it a dangerous procedure should not.

Roentgenographic Signs

1. Obscuration of the lateral border of the psoas muscle shadow and the kidney outline is considered to be a most important sign. It is the result of accumulation of pus or oedema associated with the abscess. Unfortunately, obscuration of the psoas shadow also occurs in retroperitoneal tumors, abdominal masses, appendicitis with perforation, and in many other renal lesions such as tumor, hydronephrosis, stones, polycystic disease of the kidney, etc. The loss of a clean cut kidney shadow outline suggests abscess formation. As important as this sign may be, it occurs about one to two weeks following formation of the abscess.

2. The diagnostic importance of the shadow cast by the abscess is thought by some to be most important. The opacity of the abscess produces a different appearance on the x-ray when compared with the other side.

3. Outward displacement of the kidney and inward displacement of the ureter observed stereoscopically, has been reported in a number of cases of perinephric abscesses of the lower pole of the kidney. It does not occur when the abscess is in the upper or middle portions of the kidney.

4. Curvature of the spine with concavity towards the abscess is due to psoas spasm caused by the overlaying abscess. It also occurs in trauma and infection of the spinal musculature, pleural effusion, renal stone, hydronephrosis and a variety of other conditions affecting the kidney. Although of some assistance, it is inconstant and may be misleading.

5. Displacement of the colon medially and caudally by perinephric abscess can sometimes be seen, but as a rule occurs late. This can be demonstrated by a barium enema or at times detected in a plain film in which the accumulated gas within the colon is seen in the displaced colon.

6. The displaced and fixed diaphragm occurs in the suprarenal abscess or in large pre-or retrorenal types. It is not present in the small abscess or the infrarenal type.

7. Fluoroscopic examination of the excursion of the diaphragm is of use at times, as there may be a limitation or diminution in the movement, especially on deep expiration.

Treatment

The treatment of choice is incision and drainage as soon as the diagnosis is confirmed. After the abscess has been drained, the underlying pathological processes may be treated. Occasionally, conservative palliative treatment, such as bed rest, heat, and antibiotics will effect spontaneous resolution. Cases which do respond to conservative therapy are those in which an inflammatory process or a perinephritis exists, but in which frank pus or a perinephric abscess has not as yet formed.

Summary

A correct clinical diagnosis of perinephric abscess may be arrived at by considering the history of antecedent infection of the skin, middle ear or other parts of the body; the demonstration of a tender mass in the flank on examination; the variety of roentgenographic findings, if present; and needle aspiration of the perinephrium.

Obstetrics & Gynaecology

A Review of Caesarean Section in the Misericordia Hospital

A. S. Majury, M.B., M.R.C.O.G.

Since the end of World War II there has been a notable increase in the incidence of Caesarean Section. The development of the antibiotic drugs, the great advances in anaesthesia and the availability of blood transfusion facilities have tended to make Caesarean Section a safer operation for the mother and, as a result, it is more readily resorted to when an obstetric difficulty arises. Some obstetricians feel this is a retrograde step and that in a difficult case the art of vaginal manipulation and delivery has been lost. This may be true, but often Caesarean Section is to be preferred in such a case because vaginal delivery may result in laceration or damage of the maternal soft tissues as well as serious injury to the foetus. An "ideal" incidence of Caesarean Section is impossible to define, since it will vary with circumstances—our aim should be to strive for the lowest possible Caesarean Section rate compatible with the lowest possible maternal and foetal mortality and morbidity rates.

Present Study

All Caesarean Sections performed in the Misericordia Hospital, Winnipeg, during the period 1st January 1954 to 30th September 1959 have been reviewed.

Incidence

During this period there were 9,945 deliveries of which 100 were by Caesarean Section, an incidence of 1.06 per cent. Seventy-five (75 per cent) were first Caesarean Sections and 25 (25 per cent) were repeat Caesarean Sections.

The incidence of Caesarean Section will vary with the type of obstetric practice, e.g. in British Hospitals, which deal with a high proportion of abnormal deliveries, (the normal cases being delivered at home) the incidence may be as high as 10 per cent. In the U.S.A. the incidence also tends to be high. Hess (1958) gives an incidence in New Haven, Connecticut, of 7 per cent (59 per cent of these were repeat operations) and Hall et al. (1958) in a series collected from 10 large American hospitals found an incidence of 4.46 per cent. During the 5 years 1951-1955 the incidence in the Winnipeg General Hospital was 2.45 per cent (Bradford, 1958).

It might be argued that this incidence of about one per cent at the Misericordia Hospital is too low for a hospital claiming to practice good obstetrics. In an effort to refute this argument the perinatal death rate in the Misericordia Hospital has been compared with the Winnipeg General Hospital (Table 1).

Table 1
Perinatal Death Rates (per 1,000 births) in the Winnipeg General and Misericordia Hospitals

	W.G.H.	M.H.
1956	22	36
1957	32	35
1958	27	30

While the rate at the Misericordia Hospital is slightly higher than that at the Winnipeg General Hospital, it has fallen a little each year. Admittedly the numbers are small and further study would be necessary before any conclusions can be drawn. Such a study is currently being undertaken by the combined departments of Obstetrics and Paediatrics. Perhaps a somewhat higher Caesarean Section rate would save some babies which are, at present, being lost by vaginal delivery, and this would help to lower the perinatal mortality rate.

Type of Caesarean Section

Seventy-five (75 per cent) of the Caesarean Sections were lower segment operations and 25 (25 per cent) were Classical operations (in the Winnipeg General Hospital series 18 per cent were Classical operations). Of the first Caesarean Sections 21.3 per cent were Classical operations and of the repeat Caesarean Sections 36 per cent were Classical operations.

Under the present-day conditions in a large hospital this incidence of 25 per cent for the Classical operation would seem to be unduly high. It is now generally accepted that the lower segment operation is a better and safer operation and carries much less risk of rupture of the scar during a subsequent pregnancy. There is still a definite place for the Classical operation in certain situations, but its incidence should probably be well under 10 per cent—especially if the operators are experienced obstetricians. Some operators feel that, if the patient has not been in labour, the lower uterine segment has not been formed and therefore a Classical type of operation should be done, but only very rarely should it prove impossible to reflect the peritoneum over the lower segment and incise the uterus there. Likewise some operators feel reluctant to perform the lower segment operation in the presence of a placenta praevia, but this should not be regarded as a contra-indication.

In this series one case has been included as a classical operation in which a previous classical scar ruptured at about the 34th week of pregnancy discharging the foetus into the peritoneal cavity. At operation the foetus was delivered alive, but died after a few hours, and the defect in the uterus was repaired. In another case uncontrollable uterine bleeding was encountered following a lower segment Caesarean Section for uterine inertia and a hysterectomy was performed.

Indications for Caesarean Section

In many cases there were several factors present which led to the decision to perform Caesarean Section. I have taken what seemed, according to the records, to be the principal reason for the operation and included the case under that heading. Table 2 lists the main indications and compares the present series with some others from the literature.

Table 2
Main Indications for Caesarean Section with Percentage Incidence

	Lying-in[1] Liverpool	New York	Millard[2] Fillmore	W.G.H.[3]	M.H.
Disproportion	54.3	40.0	41.6	7.6	11.0
Repeat C. S.	27.4	19.9	26.7	38.0	25.0
Placenta Praevia	7.6	7.2	3.9	15.6	27.0
Abruptio Placentae	—	5.9	4.7	1.7	3.0
Uterine Inertia	—	7.6	5.0	8.4	13.0
Malpresentation	—	3.1	3.4	3.6	13.0
Miscellaneous	—	—	—	—	8.0

1. Kerr and Moir (1949).
2. McLean et al (1950).
3. Bradford (1958).

The two most striking figures in this table are the high incidence of repeat Caesarean Section at the Winnipeg General Hospital and the high incidence of placenta praevia as an indication for Caesarean Section at the Misericordia Hospital The former is probably explained by the fact that many cases of repeat Caesarean Section are admitted to the Winnipeg General Hospital so that tubal ligation may be performed at the time of operation—of the 181 repeat sections done there 55 per cent had a concomitant tubal ligation (Bradford, 1958). The latter figure is more difficult to explain. On studying the records of these cases it would seem that sometimes there is insufficient evidence to justify a diagnosis of placenta praevia and therefore perhaps antepartum bleeding would be a more correct diagnosis. In many of these Caesarean section was performed on equivocal radiological evidence of placenta praevia without preliminary vaginal examination, where there seemed to be only a slight degree of bleeding and thus no contra-indication to a vaginal examination.

Disproportion and uterine inertia together accounted for 24 per cent of the Caesarean Sections. In all of these labour had been in progress for many hours before the decision to perform abdominal delivery was arrived at. Of the 13 cases of malpresentation eight were cases of transverse lie and five were breech presentations — four of them in primigravidae with associated uterine inertia.

Of the 75 first Caesarean Sections, eight were elective operations and were performed before the onset of labour: one multipara with a transverse lie and a bad obstetric history, one diabetic, one multipara who had had a previous vaginal repair, one occult cord prolapse, one primigravida with a transverse lie, one primigravida with disproportion and prolonged pregnancy (43 weeks) and two multiparae with history of previous difficult deliveries in whom the foetal head remained high and free. In all the remaining cases Caesarean Section was performed after labour had been in progress for some time, or as an emergency on account of vaginal bleeding.

Anaesthesia

General anaesthesia was used in 79 cases and spinal anaesthesia in 21 cases.

Blood

Table 3 shows the number of patients who received blood transfusions.

Table 3
Cases Receiving Blood Transfusions (in percentages)

	W.G.H.[1]	M.H.
Total transfused	26.9	41
One bottle	14.3	25
Two bottles	5.9	10
Three or more bottles	6.7	6

1. Bradford (1958).

There were 20 transfusions carried out during operation and 26 in the ward following operation, on some occasions as late as the seventh day of the puerperium. While the high incidence of antepartum bleeding as an indication for the Caesarean Section may partly account for the large number of blood transfusions, it is felt that many of these patients were transfused unnecessarily. Usually where only one bottle of blood is given the patient could have managed satisfactorily without it and it is now accepted that there are better and less hazardous ways of raising the haemoglobin in the puerperium than by the use of blood transfusion—Chown (1957) has repeatedly pointed out the dangers of blood transfusion in women during the reproductive years.

Morbidity

The morbidity rate in the total series was 14 per cent. In the Classical Caesarean Sections it was 28 per cent, while in the lower segment sections it was only 9.3 per cent.

There was one maternal death in the series, an incidence of one per cent. This patient, aged forty-four, had a Classical Caesarean Section for placenta praevia at the 35th week of her seventh pregnancy. The post-operative course was completely afebrile and she was discharged, apparently quite well on the eighth post operative day. Four days later she suddenly became ill and was readmitted in a moribund condition and died a few hours later. Autopsy revealed a massive generalized peritonitis.

Perinatal Mortality

Five babies were stillborn and three died in the neonatal period, making a peritoneal mortality rate of eight per cent. (Table 4).

Table 4
Showing Indication for Caesarean Section and Birth Weight of Baby in 8 Perinatal Deaths

5 Stillbirths:	
Abruptio	3 lbs. 0 ozs.
Disproportion	9 lbs. 4 ozs.
Disproportion	6 lbs. 4 ozs.
Disproportion	6 lbs. 12 ozs.
Placenta Praevia	7 lbs. 8 ozs.
3 Neonatal Deaths:	
Placenta Praevia	3 lbs. 8 ozs.
Placenta Praevia	5 lbs. 0 ozs.
Ruptured Uterus	?

This rate could have been only four per cent as, from a retrospective study of the records, it would seem there was an error of judgment in the three cases of disproportion and one case of placenta praevia which resulted in stillbirths. Earlier Caesarean Section would almost certainly have resulted in a living baby. In the other four cases it would appear that the foetal death was unavoidable.

Comment

The Caesarean Section rate in the Misericordia Hospital is lower than in most reported series. The reason for this is not apparent, but it would seem to indicate a very conservative obstetrical outlook on the part of the physicians who practice there. Double the number of Caesarean Sections could be performed without the rate becoming unduly high, yet it is difficult to see just where the indications could be widened. In view of the fact that four of the perinatal deaths might have been prevented by earlier recourse to Caesarean Section further study of the hospital perinatal mortality rate may show there were cases where vaginal delivery was inadvisedly permitted, with resultant death of the foetus, in which delivery by Caesarean Section would probably have resulted in a living baby. Thus it would be hoped that any increase in the Caesarean Section rate would result in a decrease in the total perinatal mortality rate. Nevertheless, it must be borne in mind that Caesarean Section is not necessarily the answer to every obstetrical difficulty. The fact that there was one maternal death in this series should serve as a reminder that the operation is not without risk to the mother nor, even in elective cases, does it guarantee a living baby, as the relatively high incidence of foetal hyaline membrane disease and pulmonary atelectasis following Caesarean Section, especially where the operation is performed before the onset of labour or where the foetus is premature, results in a definite foetal mortality.

Summary

1. One hundred Caesarean Sections performed in the Misericordia Hospital, Winnipeg, between 1st January 1954 and 30th September 1959 have been reviewed. Of these 75 were lower segment operations and 25 were Classical operations.

2. The incidence of Caesarean Section was 1.06 per cent.

3. The indications for Caesarean Section are listed. Repeat Caesarean Section accounts for 25 per cent of the total, placenta praevia for 27 per cent and disproportion and uterine inertia combined for 24 per cent.

4. The type of anaesthesia and number of blood transfusions are listed.

5. The maternal death rate was one per cent and the morbidity rate 14 per cent.

6. The perinatal death rate was eight per cent.

I wish to express my thanks to Miss Roma Champagne, Director of the Medical Records Department, Misericordia Hospital, for her assistance in obtaining the case records.

References

Bradford, C. R.: (1958) C.M.A.J., 78: 392.
Chown, B.: (1957) C.M.A.J., 77: 1037.
Hall, J. E., Kohl, S. G. and Schechter, H. R.: (1958) Amer. J. Obst. & Gynec., 75: 387.
Hess, O. W.: (1958) Amer. J. Obst. & Gynec., 75: 376.
Kerr, J. M. and Moir, J. C.: 1949) Operative Obstetrics. Balliere, Tindale & Cox, London, 5th Edition, page 509.
McLean, L. F., McDowell, H. C., Nichols, D. H. and Wildhack, R. H.: 1950) Amer. J. Obst. & Gynec., 60: 860.

The Perforated Uterus

Mark B. Wall, M.D.

Perforation of the uterus occurs more frequently than is generally realized, and because it is not diagnosed a good number of women who might otherwise be subjected to a laparotomy are spared this procedure. "The failure of the sound or curette to encounter resistance at the point that it normally should, as judged by the palpated size of the uterus," must lead to the consideration of a perforated uterus.

A curettage is performed as a diagnostic procedure during any stage of a woman's life, or in relation to a pregnancy during the fertile period. The cervix is dilated by dilators introduced into a firm or stenotic cervix prior to a diagnostic curettage. Perforation may occur when the resistance is overcome suddenly or when the dilator passes through the wall of the uterus in an unrecognized position. The cervix requires little or no dilatation in the postabortal or puerperal curettage, so that the danger comes from the soft, boggy uterine wall through which the sound or curette may pass readily. Occasionally a uterine malignancy or hydatid mole will produce the weakness in the uterine wall.

As soon as the operator suspects a perforation of the uterus, he should discontinue the dilatation or curettage at whatever stage he may be and try to verify his suspicions by passing a sound into the uterine cavity and, if possible, through the perforation. A sharp instrument like the curette is never used since more damage may result to the uterus and the surrounding organs. If the operator brings down a loop of bowel, or if bowel appears through the dilated cervix, the diagnosis is obvious and the bowel is carefully inspected for damage before replacing it in the abdomen. The presence of urine or feces at the cervix indicates perforation of a viscus as well as the uterus. Sudden collapse of the patient during dilatation or curettage makes the diagnosis of perforation probable.

Treatment is based on the evidence of bleeding, infection or injury to other abdominal organs. If this evidence does not appear, as is usually the

case, the patient should be kept under observation and treated symptomatically with analgesics and given a prophylactic antibiotic. In the presence of bleeding or injury to the bowel or bladder, an immediate laparotomy is necessary to repair the damage. Occasionally the site of a uterine malignancy may be perforated and a hysterectomy will prevent the further spread of malignant cells through the abdomen.

Perforation of the uterus during curettage of an incomplete abortion or of retained products after a normal delivery may be associated with infection. If the infection is spreading an immediate laparotomy is done, whether the perforation is recent or old, to provide adequate drainage and to repair the rent in the uterus. The appropriate antibiotic may then be determined and given. Perforations with no evidence of spreading infection are watched for localization of pus in the pelvis and are drained from below if necessary.

The following prophylactic measures will prevent many perforations:

1. Pelvic examination before the dilatation and curettage to make certain of the size, shape, consistency and position of the corpus uteri and its relation to the cervix.
2. Catheterization of the bladder preoperatively to prevent retrodisplacement of the uterus.
3. Adequate dilatation of the cervix to permit easy introduction of instruments into the uterus.
4. No instrument allowed to progress into the uterine cavity to a depth greater than was initially measured by the uterine sound.
5. Undue force not to be used in the removal of pieces of tissue from the uterine wall so that the myometrium is not torn through.
6. The use of an oxytoxic to thicken and firm up the myometrium before removing products of gestation.

A review of the cases of perforation of the uterus at the Misericordia General Hospital from January, 1951, to August, 1959, inclusive revealed that this complication occurred in eight cases, an incidence of 1 in 375 dilatations and curettages. Seven of the perforations occurred in the hands of gynecologists and were recognized at the time. All patients were placed under observation, given analgesics and prophylactic antibiotics and made uneventful recoveries.

Summary

1. Perforation of the uterus occurs more often than we realize.
2. Perforation of the uterus alone requires observation, analgesics and a prophylactic antibiotic.
3. Treatment must be vigorous in the presence of bowel or bladder damage, hemorrhage or advancing infection. Laparotomy, repair of the damage to the uterus and adjacent organs, antibiotics and adequate drainage are necessary.

References

1. Curtis, A. H. and Huffman, J. W.: Textbook of Gynecology, 6th ed., W. B. Saunders Co., Philadelphia, 1951, p. 218.
2. TeLinde, R. W.: Operative Gynecology, 2nd ed., J. B. Lippincott Co., Philadelphia, 1953, p. 359.

The author wishes to thank Miss Roma Champagne, Medical Librarian at Misericordia Hospital, for her assistance in preparing the data presented in this article.

Missed Abortion

Leon Rubin, M.D., M.R.C.O.G.

Definition

Missed abortion may be defined as a conception which has ceased to develop prior to the twenty-eight weeks of gestation, and in which the failure of development has been present for at least four weeks. The presence of a fetus is not essential, since many ordinary abortions occur in the absence of a fetus. A missed abortion varies primarily from an ordinary abortion in the length of time the conception is retained in the uterus after it has ceased to grow. Pregnancies which terminate after the twenty-eight weeks of pregnancy are designated as premature or term deliveries.

Diagnosis

The diagnosis of this condition depends on showing that the ovum is dead. This may be suspected if the uterus is found to be considerably smaller than would be expected from the duration of the amenorrhea, and is supported by the occurrence of a slight brown discharge of altered blood. In order to make certain, however, it is necessary to watch the patient over a period of a month, and, if at the end of this time the uterus has not grown larger, the diagnosis of missed abortion is almost certain. The patient herself may notice a diminution of the breast changes which occurred at the beginning of the pregnancy; she will not notice progressive enlargement of her abdomen nor movements of the fetus. There may also be a loss of weight. Radiological evidence of fetal death, i.e. overriding of the cranial bones, angulation of the spinal column, occur usually after intra-uterine fetal death later in pregnancy.

The biological pregnancy tests are important in confirming the diagnosis. A negative test is conclusive evidence of fetal death. The test may remain positive, however, for a short time after fetal death, as the level of urinary chorionic gonadotrophin may remain elevated for a varying length of time.

Management

It is generally agreed that conservative management is by far the safest method of handling these cases. By conservative treatment is meant that which depends on the expulsion of the uterine contents spontaneously without operative interference, or in those instances in which the expulsion followed some sort of medical induction by oxytocics

or hormones or both. Spontaneous expulsion of the uterine contents will occur, either sooner or later with very little risk to the patient. In those cases where spontaneous expulsion is delayed, the patient will undoubtedly become very anxious for active treatment to be undertaken. Surgical intervention is very dangerous as the cervix is frequently closed and rigid, the uterine wall is thin and easily perforated, and the uterine musculature is not responsive to oxytocic drugs. Also there is increased danger of hemorrhage and infection. If spontaneous expulsion is delayed, ergometrine or pitocin may be given after sensitizing the uterus with estrogens. Unfortunately this is not always effective.

Active treatment is favoured by some authors. These authors state that there are two main dangers in delaying treatment. One of these dangers is that retention of a dead fetus for an appreciable length of time may permit the development of hypofibrinogenemia with resultant severe hemorrhage. However, Battaglino and Wilson, (who favour active treatment) report only one such case and this a doubtful one, in 88 cases treated conservatively as a control series. The other main danger is serious emotional disturbance. Many of these patients are greatly disturbed because their desire to have a child has been temporarily thwarted, and also because there is something inherently distasteful in their retention of something that is dead. The above author reporting on 73 surgically treated cases performed, dilatation and curettage on 64, vaginal hysterectomy on six, and abdominal hysterectomy on three. Morbidity on these cases was no higher than in the control series. They do not intervene surgically until the fetus has been known to be dead for longer than two months.

Summing up, the management of these cases may be awkward because of the desire of the patient and her family for active intervention in evacuating the uterus. These pressures however should be resisted and almost all patients will prove to be co-operative, if the dangers of such interference are explained. Active interference should be reserved only for those rare cases in which hemorrhage occurs or serious mental disturbance appears imminent.

Pediatrics

Fibrocystic Disease of the Pancreas
A Review

Monte H. Kowall, M.D.
Department of Pediatrics, Mall Medical Group

Fibrocystic disease of the pancreas is now recognized as a well established Pediatric syndrome. The etiology is unknown. Since 1888 when Gee first described the "celiac affection," the condition remained undifferentiated from other causes of malabsorption. It was not until 1938 that Anderson suggested the separation of Pancreatic Fibrosis from the true celiac disease and emphasized the logical use of pancreatic enzyme analysis. In 1942 Farber expounded further on the etiology of this condition and related it to abnormal mucus production, applying the term mucoviscidosis. Finally in 1953 di Sant' Agnese described the abnormal sodium and chloride concentration in the sweat of Fibrocystics.

Fibrocystic disease of the pancreas could be defined as a generalized disease of infants and children whereby the secretory activity of many and perhaps all exocrine glands, mucus producing and others, is affected. What is the basic nature of this widespread disturbance? It had been postulated that mucus secreted in various parts of the body may be abnormal in its physicochemical characteristics, thus causing obstruction of pancreatic and other ducts with consequent dilatation of secretory acini and fibrosis of parenchyma. By the same token, failure to remove secretions from the bronchi may lead to obstruction of the air passages and secondary bronchopneumonia. The lesions in other organs such as the liver, were characterized by plugging of the bile ductules by concretions having the same histologic and histochemical properties as the ones in the pancreatic ducts. An abnormality of mucus was therefore probable and gives a reasonable explanation of the pulmonary, pancreatic and hepatic lesions. The sweat and parotid glands which were not mucus producing show no anatomic changes and yet were consistently affected in this disease. Current opinion leads us to believe that dysfunction of the autonomic nervous system may be responsible for such a widespread disturbance. However enzymatic abnormalities might also be responsible for the multiorgan involvement.

There was considerable evidence that cystic fibrosis of the pancreas is a genetically determined condition. When the genetics of this disease was first studied, it seemed that the disease was due to a gene which expressed itself only in the homozygote that is a perfect recessive. Consequently the incidence could be predicted with a fair degree of accuracy. However, it now seemed quite likely that fibrocystic disease resulted from the effects of gene mutation, which in turn exhibits full expression of the disease in homozygotes, but in only partial expression in some heterozygotes. The observation of Wood et al. suggested that some cases of chronic obstructive pulmonary emphysema in adults may represent formes frustes of fibrocystic disease.

Accumulated experience indicated that the disease occurred in all economic and social classes, in many racial groups, and over wide geographic areas, and in a variety of climates. The mother's condition during pregnancy seemed of little importance in inheritance of this disease. Maternal age, order of birth, and size of family had no bearing. The sexes were equally affected.

Incidence

The true incidence varied from 1:1000 to 1:10,000. Anderson estimated the incidence in New York City as 1.7 per 1,000 live births. All observers have noted a striking increase in incidence during the past six years. In 1947 there were only 40 cases at the Children's Medical Center in Boston, while in 1954 there were well over 200 cases reported. The Children's Hospital in Winnipeg reported two cases in 1954; 10 in 1955; five cases in 1956; 13 cases in 1957. The suspected reasons for this increase were presumably due to an increased awareness, a greater interest, and the easy accessibility of diagnostic procedures.

Clinical Classifications

The first and only classification was proposed by Anderson who divided the patients into the following groups:

Group 1

Meconium ileus.

Group 2

Cases with early onset of respiratory infection.

a. Small group with continuous or intermittent diarrhea in neonatal period.
b. Most patients with failure to gain on adequate diet, large, foul, formed stools and chronic respiratory infection.

Group 3

Cases with late onset especially presenting as celiac disease.

Group 4

Partial pancreatic insufficiency occurring in 13% of cases without overt clinical signs.

Actually there is no sharp separation of distinct clinical types. The course of the disease depends on age of onset, organs involved, degree or severity of involvement, and rate of progression of the lesion.

Group 1

The term meconium ileus is used to describe intestinal obstruction in the newborn caused by the inability of the intestine to propel through its lumen thick mucilaginous meconium. This type of obstruction accounted for approximately 15% of all types encountered in the neonatal period. The usual site for the obstruction was in the terminal ileum, which may be greatly distended and hypertrophied. Prior to 1945, almost all infants with meconium ileus died. This high mortality was due to late recognition of this condition, with the development of serious complications such as intestinal perforations with meconium peritonitis. With present day surgical procedures the mortality rate had dropped considerably in the newborn period. Those infants who survived surgical correction subsequently developed other manifestations of cystic fibrosis. The question arises as to whether it is possible to establish the diagnosis of meconium ileus with certainty prior to operation. Gross experienced an 80% preoperative diagnosis by history of vomiting, failure to pass meconium, abdominal distention, physical examination (palpable fecal masses in RLQ), and X-ray findings of the abdomen.

Duodenal intubation is difficult and impractical in the majority of these infants. Another screening test which is the measurement of sweat electrolytes following a thermal stress is also impractical at the age of one or two days. There is, however, a simple test to determine the presence of the abnormal mucoprotein in the bowel. This test first described by Farber resulted in a precipitation from an aqueous extract of meconium (upon the addition of 10% trichloracetic acid) from patients with meconium ileus, whereas very little or no precipitate was formed from the extract of normal meconium. In spite of this useful chemical test, the surgeon's task and responsibilities remained unaltered, inasmuch as the method of obtaining a sample of meconium would be by operative means in the majority of cases. Between 1949 and 1954, at the Children's Medical Center, 19 patients have had a Mikulicz resection and double ileostomy, the operation of choice, with a 79% survival rate. The medical management of patients recovered from surgery is essentially similar to fibrocystics unassociated with meconium ileus in the newborn period.

It was formerly believed that the patient in Group 1 represented the severest form of cystic fibrosis, and accordingly had the poorest prognosis. This is no longer justified. With successful relief of the obstruction the prognosis depends upon the severity and rate of progression of the pulmonary manifestations, rather than upon the intestinal and pancreatic dysfunction.

Group 2

Andersen found that patients in this group usually die before six months of age and many of them presented feeding problems characterized by hunger, failure to gain weight, and foul bulky stools. Usually these infants developed symptoms referable either to the gastrointestinal tract or to the respiratory system. Vomiting and diarrhea with failure to gain weight or loss of weight preceded the onset of the respiratory symptoms in some cases.

Group 3

The patients in this group possessed clinical features similar to those of true celiac disease, such as emaciation, distention of abdomen and large pale, putrefactive stools. All these patients died of advanced pulmonary disease.

Group 4

This group is thought to comprise 13% of all cases of fibrocystic disease of the pancreas. Although the most usual combination is pancreatic and pulmonary involvement, the diseases may exist with subclinical pancreatic involvement. Andersen, in a study of the pancreas in 605 routine autopsies, estimated that in 20 instances over 90% of the pancreas, and in 21 instances 20 to 90% of the pancreas was involved. In the latter group were only three patients with the clinical picture of pancreatic insufficiency. A number of observers have reported good tryptic activity in the duodenal fluid of patients who later died, with autopsy confirmation of the disease. With the sweat test it is now possible to establish a diagnosis in these patients even though there is good enzymic activity of the duodenal fluid.

Pathological Findings

The lesion in the pancreas seen most frequently in these patients was characterized by dilatation of ducts, inspissation of secretion, atrophy of acinar structures and replacement of connective tissue, leading to marked fibrosis of the organ. In most cases, obstruction appeared to begin in the acini. The islets of Langerhans were not involved. From this histological picture, interference with the production, liberation or passage of pancreatic enzymes leading to pancreatic achylia may be expected.

Hyperexpansion and atelectasis usually in combination were the commonest early findings in the lungs, and these are explained by obstructions caused by the tenacious mucopurulent material usually found in the upper respiratory tract. In the more advanced cases of the disease, widespread bronchiectatic and bronchiolectatic abscesses were found. Staph. Aureus is the offending organism. Thickening of the bronchiolar walls, replacement fibrosis in those areas of the lungs repaired after staphylococcal destruction, and diffuse bilateral acute and chronic bronchopneumonia characterized the later stages of the disease.

The liver was usually increased in size and showed fatty metamorphosis. In many cases there was an unusual form of biliary cirrhosis characterized by a lesion resembling closely that seen in the pancreas. Inspissation of secretions in the ducts and acini of the salivary glands and in the mucus glands of the trachea and bronchi, oesophagus and gall bladder occurred with regular frequency. Other evidences of general severe disturbances in nutrition and vitamin deficiency, particularly vitamin E, may be recognized elsewhere in the body.

Establishing the Diagnosis

The diagnosis of fibrocystic disease of the pancreas is essentially established as a result of laboratory investigation. The most reliable and direct method of making this diagnosis involved a quantitative examination of sweat chlorides. Abnormal sweat with characteristically increased concentration of sweat chlorides and sodium was present in 99% of patients. The group of patients studied by Di Sant' Agnese were 90 with fibrocystics disease and 151 controls, of which 135 were children six weeks to 16 years, and 16 were adults. (Figure 1).

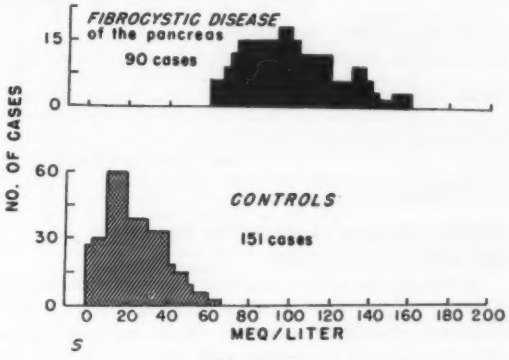


Figure 1

Concentration of chloride in the sweat of patients with fibrocystic disease and of a control group. Sweat is from midabdomen.

The range of concentration of chloride was 60 to 160 milliequivalents per liter. In patients with fibrocystic disease of the pancreas with a mean of 106; and in the controls 10 to 60 milliequivalents per liter with a mean of 32. Elevated sweat chloride did occur to a moderate degree in 2% of the general population; 14% in asthmatic children, and approximately 40% in parents of children with fibrocystic disease.

Progress had been made from the exacting earlier methods involving the collection of sweat on gauze sponges, weighing, elution, and the measurement of the sodium and chloride to the simpler and more readily available tests using a finger imprint; and still more recently the filter paper method of Webb and Gegier. There had also been a patch test developed by Gluck.

These tests depended upon the precipitation of silver chloride in a medium of silver nitrate and potassium chromate. The precipitate was then compared to a known standard. It is felt now, by many Pediatricians, that these tests were too unreliable for definite diagnosis, and any questionable case with a negative screening test should definitely have a quantitative sweat chloride estimation.

Another principal diagnostic test in fibrocystic disease was the direct measurement of the exocrine function of the pancreas. This was done by the examination of the duodenal fluid for pancreatic enzymes and viscosity. Table 1 shows the concentration of duodenal trypsin as estimated by the gelatin liquefication method. It was apparent that a number of patients with fibrocystic diseases of the pancreas had borderline values for trypsin when first seen. In 10 to 15 per cent the diagnosis cannot

be made on the basis of the trypsin concentration, as it may be normal or approach normal.

Table 1

Duodenal Fluid Trypsin in 94 Patients with FCDP
(viscosity over 3 mis. in each case)

	Duodenal Fluid, Vol. Ml.							
	1.0	.6	.2	.1	.04	.01	.005	.0025
Normal Activity	*	*	*	*	*	*	*	0
No. Patients c FCDP								
71	0	0	0	0	0	0	0	0
3	*	0	0	0	0	0	0	0
5	*	*	0	0	0	0	0	0
3	*	*	*	0	0	0	0	0
6	*	*	*	*	0	0	0	0
5	*	*	*	*	*	0	0	0
1	*	*	*	*	*	*	0	0

* Liquefaction
0 No activity

Indirect Methods of Measuring Pancreatic and Intestinal Function

Although the direct tests for pancreatic function involve a great deal of work and discomfort to the patient, one should consider these measurements essential in the diagnosis. At times it would be impossible to carry out the duodenal intubation, and under these circumstances one might resort to the so-called indirect tests of pancreatic function. The simplest procedure was the stool trypsin test which was devised as a screening test. Normal infants had significant tryptic activity in the feces. A 1:100 dilution was capable of digesting the gelatin surface from an X-ray film at 38° C in one hour. Three negative results of stool trypsin examination in an infant in the 1:5 or 1:10 dilution should be considered sufficient evidence to justify a tentative diagnosis. False negatives can occur in Hirshsprung's disease, and biliary atresia. Some patients with proved complete pancreatic insufficiency might have a positive test for stool trypsin. Johnstone explained this on the presence of gelatin liquefying bacteria in the feces. Prolonged use of antibiotics might alter the intestinal flora so that proteolytic bacteria emerge as predominant microorganisms.

Table 2 illustrated the relation between duodenal and stool trypsin. It was significant to note that stool trypsin was present in 15% of cases where trypsin was absent in the duodenal fluid. Other protein and fat absorption tests are currently in vogue.

Table 2

Comparison of Duodenal and Stool Trypsin

A. Trypsin absent in duodenal fluid

	No. Patients	Stool Trypsin Present	Stool Trypsin Absent
Infants	77	17	60
2 to 5 years	23	0	23
5 to 10 years	12	0	12
Over 10 years	1	0	1
	113	17	96

B. Trypsin present in duodenal fluid

	No. Patients	Stool Trypsin Present	Stool Trypsin Absent
Infants	105	97	8
2 to 5 years	21	15	6
5 to 10 years	3	2	1
Over 10 years	3	2	1
	132	116	16

Gelatin Absorption

Following digestion of a known amount of gelatin (1.74 gm/kg) one measured the post prandial amino acid plasma level. In patients with complete pancreatic insufficiency the ratio of the plasma glycine nitrogen after the test dose of gelatin to the fasting glycine nitrogen level was 1.8 in contrast to the ratio of 5:1 in healthy controls.

Lipiodol Absorption

A method devised by Shirkey and Silverman where iodine estimations were done on urinary specimens following the ingestion of lipiodol. In the malabsorption syndrome the value of iodine in the urine was rarely over 110 mgm.

I 131 Labelled Casein

In normal controls less than 5% of the radioactive casein was found in the feces of controls. In patients with FCDP, the average estimate is 22—5% of the ingested isotope in the feces. Balanced studies are exacting but require a co-operative lab.

Radiology

Neuhauser described two stages in the development of the pulmonary picture in FCDP (fibrocystic disease of the pancreas). The first stage was that due to widespread obstructive changes, probably caused by excessive viscid mucus, and manifesting itself as emphysema and lobar atelectasis. These mechanical disturbances were followed by the second or infective stage consisting of subacute or chronic staphylococcic bronchitis or bronchopneumonia.

Table 3 summarized the pulmonary findings in these cases.

Table 3

1. Lobar atelectasis occurred in 10% of cases of fibrocystic disease of the pancreas, usually in early infancy.
2. Usually affected the right side.
3. It indicated the severe respiratory disturbances —i.e. cough, cyanosis and respiratory distress.
4. Bronchoscopy in six out of seven cases failed to relieve obstruction.
5. Early antibiotic therapy was the best weapon.
6. Any infant who had lobar atelectasis, particularly in the right lung should be suspected of having fibrocystic disease of the pancreas.

Treatment

The general management of patients with fibrocystic disease of the pancreas involved constant medical care. Some parents required a great deal of assistance, others were satisfied with little explanation, and would follow any plan faithfully. A prepared pamphlet was used at the Children's Medical Centre in Boston to illustrate the nature of the disease and possible lines of treatment. The two most important aspects of therapy were:

1. Control or prevention of respiratory tract infections.
2. Maintenance of nutrition.

(A typical therapeutic regime is presented in Table 4).

Table 4

Nutrition

A. High caloric diet (150 to 200 Cal/kg.).
B. Satisfy hunger: 3 to 5 meals per day.
C. Avoidance of excessive fat.
D. Free use of protein hydrolysates in small infants.
E. Vitamins: water miscible in double the recommended dose.

Replacement

A. Pancreatic Enzyme extracts—
   a. Viokase powder ¼ to ½ teaspoon with each meal.
   b. Pancreatic granules ¼ to ½ teaspoon with each meal.

Antibiotics

A. Continuous treatment was recommended in most cases.

In Mild Cases ... Chlortetracycline or Oxytetracycline (5 to 15 mgm/kg day)

In Severe cases ... may add—Chloramphenicol
Streptomycin
Penicillin/aerosol.

Other drugs: should be used in severe pulmonary distress. In recent times new pulmonary techniques, such as intermittent positive pressure breathing were utilized.

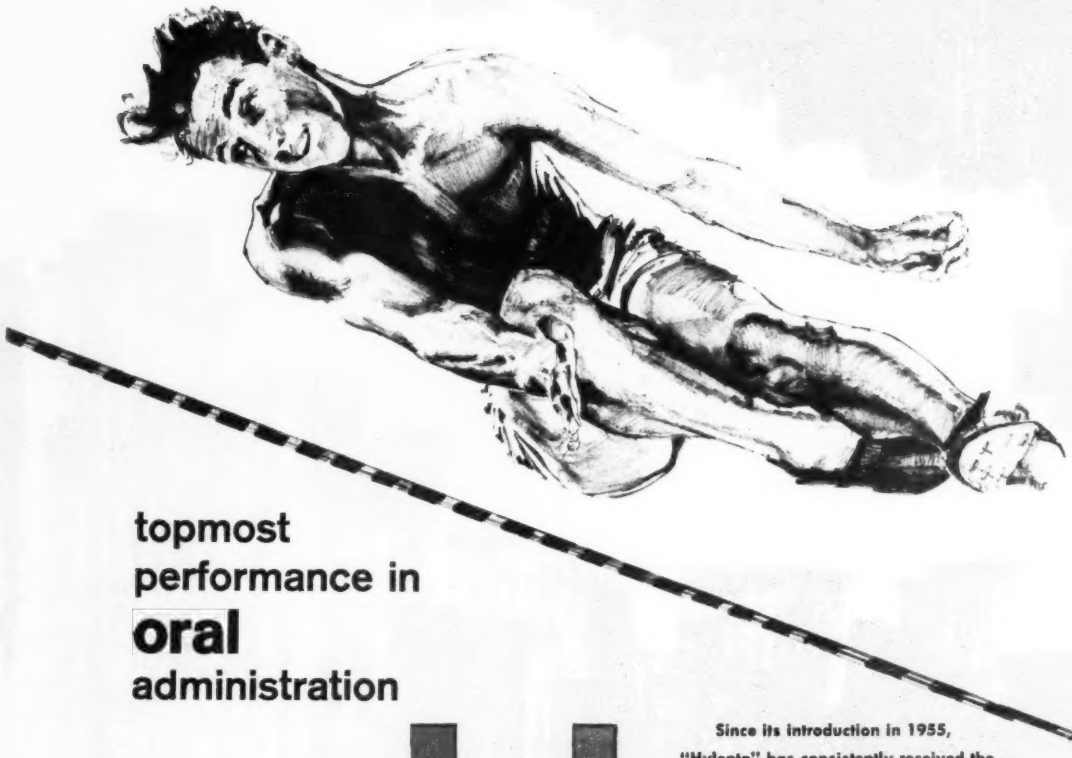
Prognosis

The prognosis for this condition is generally poor. Since the advent of broad spectrum antibiotics in 1949, the average age at death had risen from 12.8 months to 45.2 months. The prognosis to a large degree does not depend on pancreatic insufficiency but pulmonary status. To this end much research is being devoted. Detection of early cases with the onset of vigorous therapy may decrease the morbidity and subsequently affect the mortality rate.

References

1. Schwachman, H.; Leubner, H.; Catzel, P.: Musoviscidosis. Advanced Pediatrics, 7: 249, 1955.
2. Schwachman, H.; Patterson, P. R.; Laguna, J.: Studies in Pancreatic Fibrosis. Pediatrics, 4: 222, 1949.
3. Webb, B.; Giger, D.: Diagnosis of Fibrocystic Disease, "Blood, Sweat and Tears" abs. Canadian Pediatric Soc. Meeting, Winnipeg, 1957.
4. Gluk, L.: A Patch Test for Chloride in Sweat as a Simple Screening Method for Detecting Cystic Fibrosis of the Pancreas. Pediatrics, 23: 1959.
5. Kulczycki, L. L.; Craig, J. M.; Schwachman, H.: Resection of Pulmonary Lesions Associated with Cystic Fibrosis of the Pancreas. New England Journal of Med., 257: 203-208 (Aug. 1) 1957.
6. Di Sant' Agnese, P. A.: Fibrocystic Disease of the Pancreas. A Generalized Disease of Exocrine Glands. Journal of the American Medical Association, 160: 846, 1956.
7. Baxter, E. H.: Musoviscidosis. Its Clinical Aspects and Treatment. Postgraduate Medicine, 15: 334, 1954.
8. Silverman, F. N.; Shirkey, H. C.: A Fat Absorption Test Using Iodized Oil with Particular Application as a Screening Test in the Diagnosis of Fibrocystic Disease of the Pancreas. Pediatrics, 15: 143, 1955.
9. Di Sant' Agnese, P. A.: Fibrocystic Disease of the Pancreas with Normal or Partial Pancreatic Function. Current Views on Pathogenesis and Diagnosis. Pediatrics, 15: 1 683, 1955.
10. Wood, J. A. et al.: A Comparison of Sweat Chloride and Intestinal Fat Absorption in Chronic Obstructive Pulmonary Emphysema and Fibrocystic Disease of the Pancreas. New England Journal of Medicine, 19: 260, 1959.

Beginning with the May 1960 issue, the Manitoba Medical Review will devote a section to questions and answers dealing with problems of medical economics, Manitoba Medical Service and Association affairs. Members of the profession are urged to avail themselves of this forum.



topmost
performance in
oral
administration

Since its introduction in 1955, "Hylenta" has consistently received the most favorable reception by the medical profession. There MUST be a reason.

HYLENTA

FORTIS AR* TABLETS
NO. 891

1 million I.U.
penicillin G potassium

Also available

"Hylenta" Mitis CD**
(250,000 I.U.), No. 890

"Hylenta" CD**
(500,000 I.U.), No. 888

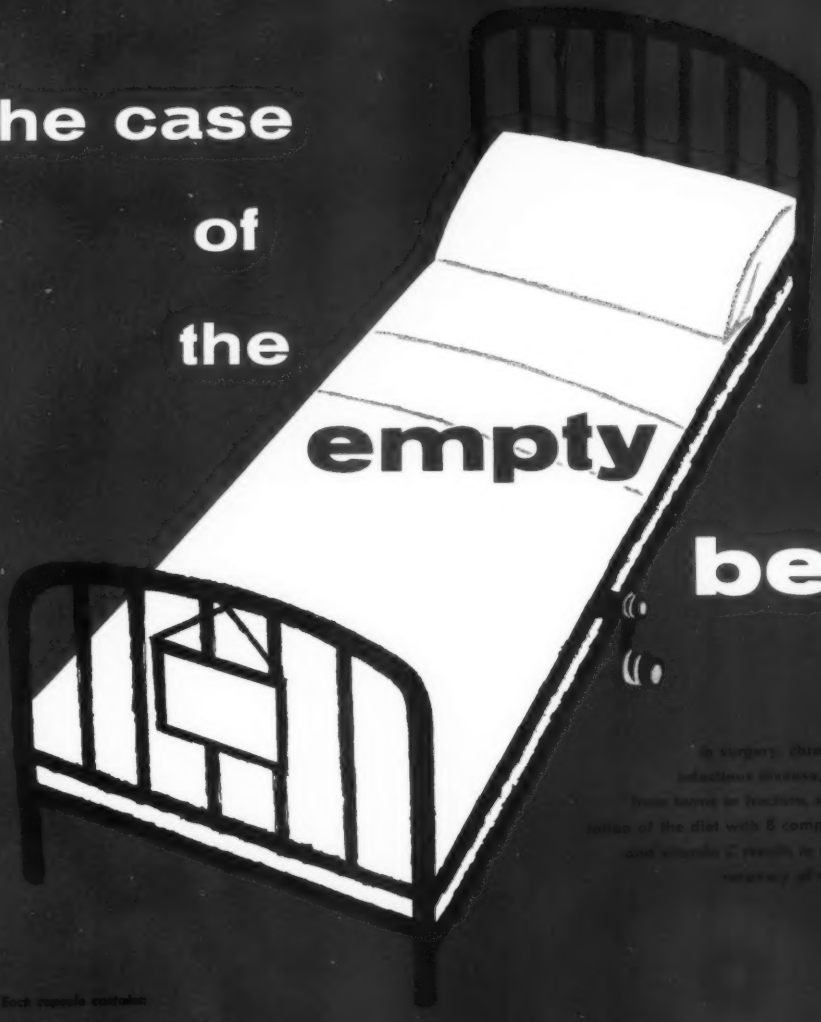
Ayerst

Ayerst, McKenna & Harrison Limited—Montreal

*Acid Resistant coating
**Controlled Disintegration base

H-5

the case of the empty bed



In surgery, chronic illness, infectious diseases, or trauma from burns or fracture, supplementation of the diet with B complex factors and vitamin C results in more rapid recovery of the patient.

Each capsule contains:

Vitamin B$_{12}$	10.0 mcgm.
Thiamine	25.0 mg.
Riboflavin	12.5 mg.
Niacinamide	50.0 mg.
Pyridoxine	1.0 mg.
Calcium d-Pantothenate	10.0 mg.
Vitamin C	250.0 mg.

Dose: one to three capsules daily.

"Beminal" with C Fortis
No. 817

Ayerst

Now available: ELIXIR "BEMINAL" FORTIS No. 925
nutritional tonic with taste appeal

Ayerst, McKenna & Harrison Limited—Montreal

8-1

ANNUAL MEETING

INTERNATIONAL COLLEGE OF SURGEONS

CANADIAN AND UNITED STATES SECTIONS

Marlborough Hotel, Winnipeg

September 28th and 29th, 1960

Immediately following the Manitoba Medical Association Meeting
September 26th and 27th

Guest Speakers Participating in the Program:

APPLEBY, Lyon H., F.R.C.S. (Eng.), F.R.C.S. (C), F.A.C.S., F.I.C.S., Surgeon-in-Chief, St. Paul's Hospital, Vancouver; Cons. Surgeon, Children's Hospital; Clin. Associate Professor (Surg.), University of British Columbia.

BACON, Harry E., F.A.C.S., F.R.S.M. (Hon.), F.I.C.S. (Hon.), Professor and Head of Department of Proctology, Temple University Medical Centre, Philadelphia, Pa.

COMPERE, Edward L., F.A.C.S., F.I.C.S. (Hon.), Professor and Chairman, Department of Orthopedic Surgery, Northwestern University Medical School, Chicago, Ill.; Senior Consultant, Orthopedic Surgery, Veterans Administration Research Hospital, Chicago, Ill.

DE LOS REYES, J. M., F.A.C.S., Sr. Staff, California Hospital; Staff, Santa Monica Hospital and Queen of Angels Hospital (LA); Cons. Physicians and Surgeons and Behrens Memorial Hospital (Glendale); St. Francis Hospital (Lynwood); Norwalk State Hospital (Norwalk); French and California Babies & Children's Hospitals; Presbyterian Hosp.-Olmsted Memorial (LA).

GROSSMAN, Arnold A., F.I.C.S., F.R.C.S. (C), Assistant Otolaryngologist, Montreal General Hospital; Demonstrator, Department of Otolaryngology, McGill University.

HOLLENDER, Abraham R., F.A.C.S., Professor Emeritus, Illinois; Clinical Professor, Miami; Cons. Mt. Sinai, St. Francis & Miami Beach Hospitals; and Variety Children's Hospital (Miami).

JACKSON, Arnold S., Chief of Staff — Methodist Hospital, Madison, Wisconsin.

LEDERER, Francis L., F.A.C.S., F.I.C.S. (Hon.), Professor and Dept. Head, Illinois; Att. Michael Reese, Grant, Mt. Sinai and Columbus Hospitals; Ch. Att. Research and Educational Hospitals; Ch. Svc. Illinois Eye and Ear Infirmary; Consultant V.A. Hospital (Hines) and U.S.N. Hospital (Great Lakes); Chicago, Illinois.

O'DONAGHUE, John B., Clinical Professor, Loyola; Professor, Cook County Graduate School; Att. Cook County and Mercy Hospitals, Chicago, Little Company of Mary Hospital.

OWENS, Neal, Clinical Professor, Tulane; Department Head E.E.N.T. Hospital; Staff, Illinois Central, Sara Mayo & Crippled Children's Hospitals; Associate Surgeon and Consultant Plastic Surgery, Touro Infirmary; Vis. Charity Hospital of La.; Cons. U.S.P.H.S. Hospital; New Orleans, La.

ROSEN, Samuel, M.D., D.A.B., F.I.C.S. (Hon.), Consulting Otologist, Department of Otolaryngology, and Chief of the Stapes Mobilization Clinic, Mount Sinai Hospital, New York City; Associate Clinical Professor of Otolaryngology, Columbia University; member, American Otological Society, American Otorhinolaryngology Society, American Academy of Ophthalmology and Otolaryngology, New York Academy of Medicine and American Medical Society of Vienna (Hon.).

SADOVE, Max, Diplomate of the American Board of Anesthesiology; Professor of Surgery (Anesth.), U. of Illinois College of Medicine, Chicago; Head of Department of Anesthesiology, U. of Illinois Research and Educational Hospitals; Director of Anesthesiology, V.A. Hospital, Hines, Ill., and West Side V.A. Hospital, Chicago, Illinois.

STREAN, George J., F.I.C.S., F.R.C.O.G., F.A.C.S., M.R.C.O.G., F.R.C.S. (C), Director, Department of Obstetrics and Gynecology, Jewish General Hospital, Montreal, P.Q.; Assistant Professor of Obstetrics and Gynecology, McGill University, Montreal.

THOMPSON, Gershom J., F.I.C.S. (Hon.), Professor and Head, Dept. of Urology, University of Minnesota, School of Medicine, Mayo Foundation, Rochester, Minn.; President of the Staff, Mayo Clinic, Rochester, Minn.

THOREK, Philip, F.A.C.S., F.I.C.S., Associate Professor of Surgery, University of Illinois College of Medicine and Cook County Graduate Schools of Medicine, Chicago.

TOTTEN, Harold P., M.D., M.Sc. (Surgery); Diplomate of American Board of Surgery; Associate Clinical Professor of Surgery, College of Medical Evangelists, Philadelphia, Pa.

In addition there will be Sectional Meetings in the following specialties — Obstetrics and Gynecology, Urology, Orthopedics, Anesthesia and E.E.N.T.

All doctors are invited to attend.

Registration Fee $5.00.

RESIDENT INTERNES AND MEDICAL STUDENTS — NO REGISTRATION FEES.

in HYPERTENSION and DIABETES

REDUCE the hazard of HEMORRHAGE

"CERUTIN"

Rutin with Vitamin C

FOR THE PREVENTION OF VASCULAR ACCIDENTS
ASSOCIATED WITH INCREASED CAPILLARY FRAGILITY

"CERUTIN"
Tablet No. 388 Trade

Rutin N.F.	20 mg.
Vitamin C.	25 mg.

"CERUTIN" R 60
Tablet No. 389 Trade

Rutin N.F.	60 mg.
Vitamin C.	120 mg.

DOSAGE: One tablet 3 times daily.

Packaged in bottles of 40 and 100.

"Although no significant improvement in vision or decrease in retinopathy was observed in diabetes after rutin therapy, it may be significant that no loss of vision or increase in retinopathy occurred during a period of 10 to 12 months' treatment".[1]

The results of treatment with Cerutin are not dramatic. It is necessary to adopt a long range viewpoint. Cerutin should be prescribed for every patient with hypertension and diabetes, with the hope that capillary rupture in retina and brain may be avoided or postponed.

1. The Effective Use of Rutin. Donegan and Thomas, Am. J. Ophthal., 31:671, 1948.

6-6

Charles E. Frosst & Co.
MONTREAL CANADA

The "Misery"

Having become accustomed to acting host to hospital issues, the Review welcomes without a trace of self-consciousness and with all the grace it can muster, the hospital of the month — Misericordia General. With a relaxed and informal gesture, it bids the honored guest to enter and unpack his literary baggage.

To the reader who has grown somewhat tired of reverential references to research and teaching in the introductions of the previous hospital issues, there may be a measure of relief in the assurance that this aspect of hospital activity will not be belabored in this one. It will be mentioned, of course that although lacking official affiliation with the Medical College, the Misericordia, like all good hospitals, carries out its own "home-made" efficient program of teaching in the form of lectures, ward rounds and clinical demonstrations for its internes and staff. It will be emphasized, however, that the reader, having saturated his mind with comment about medical education should give more than passing thought to that other unglamorized, yet essential function of a hospital — the care of the patient. The reader will be reminded that somewhere far removed from the conference rooms, committee chambers, and record offices, there is the sick man about whom all the activity revolves. This man demands constant and competent care, and it is the quality of this care that determines whether the hospital lives up to its main function. There is little doubt that the Misericordia will pass this acid test of excellence with flying colors.

The Misericordia General is often referred to by its Staff as the "Misery." This is not an expression of derogation, but an affectionate abbreviation. There is nothing about the Misericordia to suggest misery, except possibly the latter's proverbial love of company. In this case, the company is the jolly one of a faithful staff imbued with the spirit of service. Ed.

Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Guest Editorial

Misericordia General Hospital

The Medical Staff of the Misericordia General Hospital gladly submits the collection of papers for this issue of the Manitoba Medical Review, in response to our editor's kind invitation.

The story of our hospital is an interesting one, going back to the year 1845 when the Sisters of Misericordia were established in Montreal. At the request of Archbishop Langevin, the Order was invited to come to Winnipeg to assist in the moral re-establishment of unmarried mothers and "to receive, nurse and bring up the poor forlorn children of our civil society."

The Sisters accepted this call and arrived in Winnipeg in 1898. Within 2 years, they established a modest building on the present site for the provision of obstetrical care. By 1907 this had developed into a small Maternity Hospital. Because of the impact which these sisters had made upon the community they were urged by the public and local doctors to provide a full hospital service. Thus in 1916 MISERICORDIA became a General Hospital.

Last year, the Sisters of Misericordia celebrated their 60th year of service in Manitoba. The work of the Sisters in the field of obstetrical care is well known across the North American Continent and as far away as Africa. Their contribution towards Sanatorium and General Hospital care is relatively recent in history, and is now becoming more widely known. Although the Sisters have never lodged a public appeal for funds, they have succeeded in winning the support of many benefactors who have made possible an expansion of the original buildings, culminating with the vast Cornish Wing which was opened in 1957.

The addition of this Cornish Wing not only improved our outward appearance, but it greatly added to our ability to provide better patient care. The matter of settling in did reveal certain features which failed to facilitate our busy schedule, but in the main we have been immensely proud of our new wards, the 12-room operating suite, and spacious recovery area. More recently the opening of an intensive care unit of 14 beds will add greatly to the standards of surgical nursing and plans are underway to apply this principle on other services. With the continuing shortage of nursing personnel this may eventually come about by some measure of self-care among those in convalescence.

During 1958, a new Department of Laboratories occupying over 7,000 square feet was established on the site formerly occupied by the Operating Rooms.

1959 was even more eventful. Shortly after the new year, the Department of Physical Medicine and Rehabilitation was established with one Physiotherapist under a Director. Three Physiotherapists are now fully occupied and a fourth is to be added shortly. A full range of care, short of hydro-therapy is now being provided.

On the 6th floor, the new Department of Psychiatry has just been celebrating its first anniversary. This ward of 17 beds is running at full occupancy. In addition to these new improvements, the Department of Radiology has been completely rebuilt, and two other floors offering standard ward care have been remodelled and decorated.

Our chief concern is that we, in common with others, are faced with a shortage of trained personnel, and hospital services can never expand without them. As a hospital grows it does so in two ways. There is first the physical plant itself with all its architectural features designed to facilitate patient care, and then there is the invisible soul of the institution, made up of personnel trained in their specific fields, performing jobs efficiently in a spirit of unity, and with a sense of belonging to one large team. Both aspects are very important, and both must grow in proportion to each other. MISERICORDIA has been expanding from both aspects, largely due to the active interest and leadership shown by our Medical Staff Organization.

The staff was organized first in 1953 and has almost tripled in number since that time. A very close bond now exists between Medical Staff and Administration. But with the rapidly changing hospital scene now being experienced by all of us in Manitoba, we stand with many others at a cross road, wondering how best we can serve the public within the next 10 years. Because of the strength which now exists, we look forward with confidence to the many interesting years which lie ahead.

John Scatliff, M.D., D.P.H.,
Medical Director,
Misericordia Hospital.

in oral penicillin therapy

the speed of action you want

the reliability you need

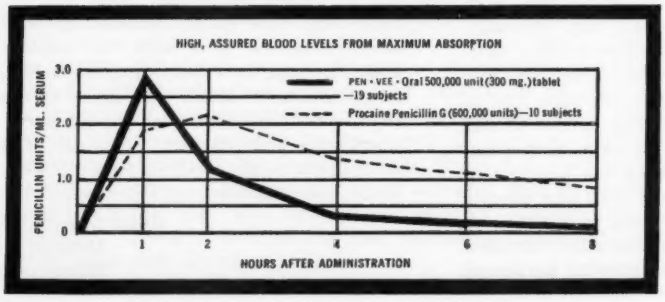
PEN·VEE·*Oral* may be prescribed for all infections responsive to oral penicillin ... and even many usually treated with parenteral penicillin.

PEN·VEE·*Oral* Tablets, Crystalline Penicillin V, 125 mg. (200,000 I.U.) and 300 mg. (500,000 I.U.) – bottles of 12 and 100.

PEN·VEE *Suspension,* Benzathine Penicillin V Oral Suspension, 90 mg. (150,000 I.U.) and 180 mg. (300,000 I.U.) per 5 cc. teaspoonful – bottles of 2 fluid ounces.

PEN·VEE*·*Oral*

Tablets and Suspension



℞ Available on prescription only

*Reg. Trade Mark Patented 1953

Wyeth

Medical History

A Case History from the Spacious Days

Athol Gordon, M.D.

It is indeed an honour and a privilege to be invited to contribute a paper to the Medical Review and to the Misericordia Hospital number of that Journal. To be in practice today is an experience both stimulating and frustrating: Stimulating in that we are armed as never before against disaster and disease; frustrating in that the choice of weapons is so wide, that we may well fear that we are perhaps destined to succeed or fail by the presence or absence of a single hydroxyl group, an atom of iodine, one of the 17 keto-steroids, or a molecule of pantothenic acid. The clatter of the arrows in our modern quiver may well distract us. If we withdraw to our library to consult our journals we must wade through a mass of advertising only to find that it is present in something of the order of three to one as regards actual medical or surgical material. The picture is one of the doctor finding himself in the midst of a rapidly expanding medical universe, glad to be there, but well nigh distraught by the effort to keep apace of it. He must be increasingly thankful for the torrent of medical abstracts from which he can fish the occasional appropriate trout.

In spacious days of Elizabeth the First and long before, the value of the case history was passed from teacher to student; but in those days the teacher realizing the value of the history took it himself and did not delegate the duty to a pupil. Dr. Robert Record in his book The Urinal of Physic says:

". . . so this judicial of urines, tho it be a thing highly to be regarded, yet if it be used rashly without communication of other signs known therewith, might cause, as it does often, some error to be in the sentence of the physician tho he were right excellently learned; not so much by the ignorance of the physician as by lack of knowledge in the patient, which should instruct the physician in such questions as need to demand of him; and not to look that the physician should tell him all at the first sight, more like a god than a man. *So if there be any physician so arrogant that he will take upon him to tell all things alone, and will not hear the patient speak, specially not knowing the party before . . . such a man is unworthy to be called a physician.*"

Perhaps the good doctor at some time was made the victim of a jest; or perhaps some patient sending a specimen was anxious to get more than full value for his money. I think I can detect a note of wounded vanity where he writes:

"And likewise I shall exhort all men, not to mock and jest with any physician (as some lightwits do) tempting them with beast's stale instead of men's urine, or bringing to them men's water for women's and such other like things."

In the year 1588, just after the defeat of the Spanish Armada there appeared a translation of the medical writings of Doctor Franciscus Arceus. It was made by Doctor John Reade, Surgeon, and Wycliffe had already given the Bible to the people of England in their own tongue. Doctor Reade under a like motivation was making the works of Franciscus Arceus and his predecessors available to the increasing medical fraternity not versed in the Latin tongue. This translation is full of interesting medical lore showing clearly that within the framework of his knowledge the Physician and Surgeon was a keen observer and a good practitioner.

Here is the story of an abscess in the abdominal wall:

". . . and the imposthume was now ripe and opened of us. It happened on a certain day in the morning when we had made clean the wound, by chance we saw a hull in the very ulcer; but I, thinking the same to have been mingled in with the lints of the day before, and so to have stricken fast to the flesh, did cause it to be taken away with the pullets, and at last the stalk with the ear did most easily follow as I drew it. But always the physicians be amazed at the strangeness of the thing, and also others that stood by, we could not guess what the matter should mean, until that old man be moved with our talk, lifted up his head, and seeing the ear sticking in the forceps, said unto me:

"I myself did thrust it into my yard a year and a half since," and so declared all the whole matter, how it was done, and the times in which it was altogether thrust in. In which place great admiration came upon us, and most especial occasion to praise the Divine Providence; for thus God provided for the man that the skin of the bladder should be broken by little and little, and so expel the ear from the bladder, being broken to the flesh, and that was hurt; and after, that the skin of the bladder should come together; and the flesh being impostumated should ripen, and the impostumation should be ripe and cured, the ear should come forth, and that, the length within very few days, the old man should be delivered, for he was thoroughly cured."

Medical science has travelled a long road since those days. Increasing knowledge has brought sophistication and cynicism. Medicine has let go the hand of Philosophy to embrace the whole body of Science. We do not yet know all, and the physician who walks with God in the quiet hours will approach that ideal state known as aequanimity, the goal of the entire human race. So let us not

FORTABEX

Oral vitamin B-C therapy for all body need

FORMULA PER TABLET

Thiamin (B_1)	25 mg.
Riboflavin (B_2)	10 mg.
Pyridoxin (B_6)	6 mg.
Niacinamide	100 mg.
d-Panthenol	10 mg.
Vitamin B_{12}	10 mcg.
Ascorbic acid	150 mg.

FORTABEX is indeed a well balanced therapy: to correct inadequate *nutrition,* to buttress body defence against *infection,* to maintain psychosomatic equilibrium in periods of *stress* and to shorten convalescence following surgery.

ROUGIER MONTREAL

Nutritional supplement and psycho-tranquillizer in mild neuroses

FORTABEX-TRAN

(Fortabex + tranquillizer)

smile the smile of smug self satisfaction as we watch our Elizabethan confreres filled with "admiration" at that which we have a full set of modern phrases to describe today. A century from now if there is a medical world left, it may be smiling at us as we shroud the common cold and the abnormal behaviour pattern in the voluminous robes of our own ignorance. The idea of escape may well flit across the medical mind in this age of speed, and there is a wondrous calm backwater in the stream of medical progress which may still be fished with assurance of a good catch. The equipment is simple; the lure of observation, the line of time, and the rod of purpose. The pool bears a sign on its bank which reads: "Welcome Fishermen . . . No Closed Season . . . Don't Throw the Little Ones Back . . . Come Again to Case History Pool . . . Take Home a Good Catch."

Thank you Doctor Arceus — Thank you Doctor Reade.

Reference

"A most excellent and compendious method of curing wounds in the head and in other parts of the body with other precepts of art, practiced and written by that famous man, Franciscus Arceus, doctor in Physic & Surgery and translated into English by John Reade, Surgeon."

Imprinted at London by Thomas East for Thomas Cadman, 1588.

The book was the property of Edward Paige, Surgeon.

Winnipeg Medical Society

Report of Nominating Committee

The following report was presented to and accepted by the Council of the Winnipeg Medical Society on February 15th, 1960. Prior to that time all candidates agreed to allow their names to be placed on the ballot for election at the Annual Meeting in 1960. The Nominating Committee is pleased to present the following report:

Vice-President:
Dr. D. L. Kippen
Dr. J. T. MacDougall

Secretary:
Dr. T. W. Fyles
Dr. J. R. Mitchell

Treasurer:
Dr. D. J. Hastings
Dr. A. B. Houston

Trustee:
Dr. W. J. Hart
Dr. J. W. Whiteford

Honorary Life Members:
Dr. C. E. Corrigan
Dr. R. W. Richardson.

Book Review

The Arterial Wall. Edited by A. I. Lansing, A.B., Ph.D., 259 pages. The Williams and Wilkins Co., Baltimore, 1959. Price $7.50.

This book concerns the aging, structure and chemistry of the arterial wall. Contributors of the ten chapters covering the several aspects of this problem were C. A. Woerner, B. W. Zweifach, W. F. H. M. Mommaerts, W. Batchelor and C. Levene, A. I. Lansing, J. E. Kirk, C. H. Barows, Jr., and B. F. Chow, R. J. Boucek and Nancy L. Noble, A. L. Lehninger and finally, A. Dury who wrote the summation. It was intended to gather together in one place the bulk of information available which might be of help in gaining a better understanding of the processes and results of aging in the arterial wall. It is admitted that no definitive relationship of any observations to chronologic age of the organism was shown and that, in fact, one could hardly have expected any to be shown, since there is as yet no agreement on either the concept or definition of what constitutes "biologic aging." It is obvious, as one reads that arteriosclerosis and atherosclerosis are prominent in the minds of many, if not all, of the authors. Except for the material presented in the excellent chapter on vascular endothelium by Zweifach, the book is largely concerned with the properties of the larger vessels of the order of the coronary and cerebral arteries and the aorta.

The kind of information presented can be conveniently indicated by the titles of the chapters as follows: the vaso vasorum of arteries, their demonstration and distribution; structure and behaviour of vascular endothelium; perspectives in the study of arterial muscle; collagen and ground substance; elastic tissue; mucopolysaccharides of arterial tissue; studies on enzymes in arterial tissues; lipid metabolism of connective tissues as related to vascular aging; the metabolism of the arterial wall; summation. Except for the first two chapters the approach to the arterial wall is almost entirely biochemical in nature. Discussion of the pharmacology and other physiological aspects of the arteries has been largely omitted or referred to in other literature. The aim has been to appraise the knowledge which might pertain to aging of arteries. However, because so little is known about aging and an understanding of the normal physiology of the vessel wall is obviously a prerequisite to the study of aging, the effort has resulted in a critical review of work, largely biochemical in substance, on the basic physiology of arteries. The extensive bibliographies allow easy entry into the literature.

It quickly becomes apparent how little information has been obtained from studies on the arterial wall itself. A great deal of the knowledge provided on elastic tissue or metabolism of connective tissues for example has been gained from investigation of

whenever there is inflammation, swelling, pain

VARIDASE®
STREPTOKINASE-STREPTODORNASE LEDERLE
BUCCAL *Tablets*

conditions for a fast comeback...

as in acute hemorrhoids...

SUNDAY, 9 A.M.: VARIDASE for painful thrombotic hemorrhoid. 2:30 P.M.: pain greatly reduced, less swelling and inflammation.

MONDAY: size down to small tab; acute inflammation disappeared.*

VARIDASE activates natural fibrinolytic factors, to limit undesirable inflammatory response and speed healing.

Dramatic reduction of pain is often the first sign of improvement; swelling and redness rapidly diminish. Drugs and natural regenerative factors readily penetrate the inflammatory barrier to effect total remission faster . . . in trauma or infection.

VARIDASE Buccal Tablets contain:
10,000 Units Streptokinase, 2,500 Units Streptodornase.
Supplied: Boxes of 12 and 100 tablets

*Peterman, R. A.: Clinical report cited with permission.

Lederle

CYANAMID OF CANADA LIMITED *Montreal*

the properties of these tissues in other sites and the relevance of this material to the arterial wall is discussed. A striking example of our paucity of facts concerning the arterial wall is provided in the chapter on perspectives in the study of arterial muscle. This chapter is sixty-six pages long, but only twelve of these deal with smooth muscle, and even this is not restricted to arterial smooth muscle, but treats of smooth muscle in general. The bulk of the chapter consists of an excellent and stimulating appraisal of work on striated muscle in the hope that the information already gained in this field may in some respects be applicable to arterial smooth muscle or, at least, give leads in the investigation of its structure, function and chemical characteristics.

The chapters are for the most part well-written and clear. Certain of them, such as that on endothelium, would be of interest to a wide circle of readers. However, this book will be of particular value to those already engaged in or about to be engaged in research on arteries. Even biochemical studies of tissues seem to have become rather specialized and in bringing together current ideas and facts about the properties and functions of all the various tissues comprising the arterial wall a worthwhile service has been rendered in providing a more complete picture for those investigating some small facet of the arterial system. Much food for thought is provided and many opportunities indicated for anyone contemplating work in this field. Scientists interested in the biochemistry of the common tissues generally and any who are concerned with skeletal muscle will also find this volume very useful.

P. G.

Abstracts from the Literature

The Solitary Circumscribed Pulmonary Lesion Due to Bronchogenic Carcinoma. Vance, J. W., Good, C. A., Hodgson, C. H., Kirklin, J. W. and Gage, R. P. Diseases of the Chest. 36: September, 1959.

The significance of the solitary pulmonary nodule has received increased emphasis in recent years in view of the demonstrated fact that a sizable proportion of these circumscribed lesions are malignant. It is assumed that a bronchogenic carcinoma which presents in this manner is more amenable to complete surgical extirpation and thus will have a better prognosis.

In the eleven year period between 1944 and 1954, there were 94 surgically treated cases of bronchogenic carcinoma which had presented as a solitary circumscribed pulmonary lesion. Males predominated and only two patients were under 40 years of age. Symptoms were present in about half the cases. Not one of the lesions showed roentgenographic evidence of calcification. Fifty-two of the lesions were greater than 4.0 cm. in diameter, and none was less than 1.0 cm. All patients were thought to be operable on clinical grounds.

Nine of the 94 patients were found to be inoperable at the time of exploration because of extensive mediastinal metastases. The resectability rate was thus 90 per cent. In another three patients, it was obvious at operation that not all the diseased tissue could be excised, and palliative resections were done. This left 82 patients who had resection for what appeared to be all of the evident carcinoma, giving a "resection-for-cure" rate of 87 per cent. Large cell carcinoma, adenocarcinoma and squamous cell carcinoma each accounted for about 30 per cent of the 94 lesions.

All but one of the patients were followed and were eligible for calculation of three-year survival rates; the one exception was last known to be living two years after pneumonectomy. None of the nine patients on whom only an exploratory thoracotomy was performed survived for three years, and seven were dead within the first year. All three patients who had received a palliative resection were dead within the first year. Of the 82 patients who underwent "resection-for-cure," five died in the hospital, one was only followed for two years, and 34 of the 76 remaining patients survived for three years or longer following operation. This is a three year survival rate of 44.7 per cent. The three year survival rate of the entire series was 36.6 per cent.

Patients with no thoracic symptoms had a survival rate 15% higher than those with symptoms; the three year survival rate was also lower if the sedimentation rate was greater than 30 mm. or if there were positive cytological findings.

The above data were compared with those from the same institution for a series of 1,577 cases of bronchogenic carcinoma presenting in the usual fashions. In this latter group, thoracotomy was only performed in half the cases, and the resection-for-cure rate of the operated cases was only 49.8 per cent and the operative mortality was 13.6 per cent. The three year survival rate of the patients undergoing "resection-for-cure" was over 40%, almost identical with that in the patients with solitary circumscribed malignancies.

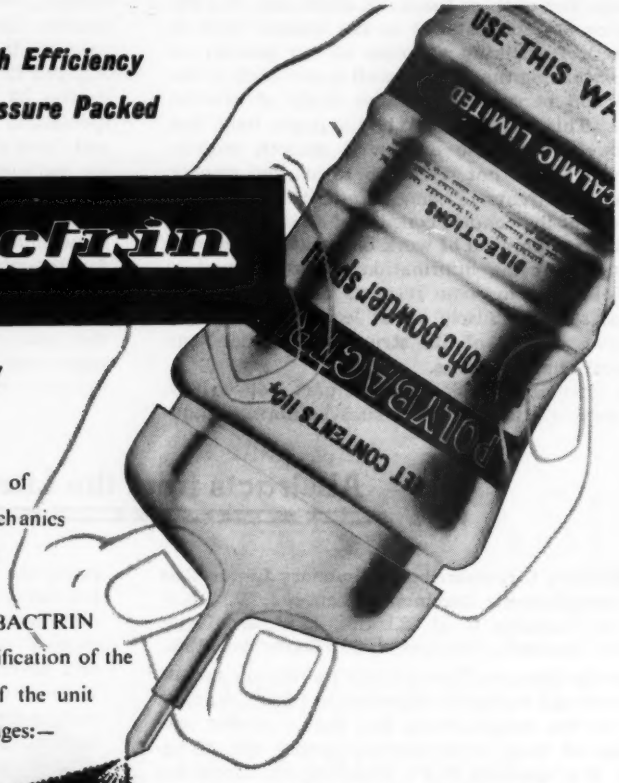
The higher resectability rate for bronchogenic carcinoma presenting as a pulmonary nodule (90% as compared to 24%) increases the over-all survival rate for patients with this form of bronchogenic carcinoma as compared with patients who have other forms of bronchogenic carcinoma.

G. A. Lillington.

NEW

High Efficiency
Pressure Packed

Polybactrin



Antibiotic Powder Spray

This new presentation of POLYBACTRIN is the result of intensive research into the mechanics of pressure packing antibiotics in powder form. While the formula and potency of POLYBACTRIN remain unchanged, radical modification of the valve mechanism and design of the unit provides the following advantages:—

NEW — positive directional, wide-angle spray gives wider coverage of the area under treatment.

NEW — all metal container provides additional safety

NEW! — special valve eliminates impacting of powder and clogging of nozzle

A PRODUCT OF CALMIC RESEARCH

The use of Polybactrin during surgical procedures is a positive method of inhibiting any wound pathogens which may become implanted in the tissues.

INDICATIONS: Prophylactic—treatment of Burns—general surgery Gynaecological operations—orthopaedic surgery etc.

FORMULA: Powder content 1.5g.
Each gramme contains:

Neomycin Sulphate	500 mg.
Polymyxin B Sulphate	100,000 units
Zinc Bacitracin	25,000 units

Pressurised with inert chlorofluorohydrocarbon propellants.

NET CONTENTS 110g.

CALMIC LIMITED
220 BAY STREET, TORONTO
Crewe and London, England
Sydney, Australia

Broncholithiasis: Moersch, Herman, J. and Schmidt, Herbert, W. Annals of Otology, Rhinology and Laryngology. 68: 548, June 1959.

Broncholithiasis (bronchial stone) is usually the result of erosion and protrusion of calcified hilar and paratracheal nodes into the tracheobronchial tree. Rarely it may be due to calcification about a foreign body or to ossification in bronchial cartilage with subsequent erosion and sequestration of the calcified material into the bronchial lumen. In 99 proven cases studied at the Mayo Clinic, tuberculosis was proven by bacteriological means in only three cases, and in one third of cases the tuberculin skin reaction was negative.

The symptoms produced by broncholithiasis depend on the size and location of the bronchial stone. In five cases, the stone was so small that it did not produce bronchial obstruction of sufficient degree to cause symptoms. Cough was present in 80 per cent of the patients, dry in the early stages but often productive of mucopurulent sputum later. In half the cases there were episodes of bronchial infection associated with chills and fever. These recurrent episodes tended to last for several days and often subsided with the expectoration of malodorous purulent material. Hemoptysis, usually minor in degree, was present in two-thirds of the cases. Almost half the patients gave a history of having expectorated one or more broncholiths, and one patient stated he had coughed up 103 stones. Some complained of pain and wheezing.

Physical findings vary with the degree of bronchial obstruction and are never diagnostic. Roentgenologic findings are usually present, and the most characteristic finding is the presence of an area of collapsed lung tissue with dense calcification at its apex. The roentgen findings are often much less specific than this however. Bronchoscopy was performed in 87 cases; broncholiths were found in 38 of the patients and were removed successfully in 33 instances. In 35 patients, bronchoscopy failed to reveal a broncholith but showed other evidence of bronchial disease. The broncholiths were twice as common in the right lung as in the left.

Diagnosis was established only at exploratory thoracotomy in about a third of the cases. Surgical treatment with resection of lung tissue was performed in 38 patients. In some instances, the broncholith had been removed or expectorated previously but resection was required because of bronchial stricture and bronchiectasis. The authors emphasize that the presence of a broncholith within the lung does not exclude the possibility of other associated pulmonary disease such as carcinoma or tuberculosis.

G. A. Lillington.

Surgical Treatment of Stage I Cancer of the Cervix. A. Brunschwig. Cancer: Jan.-Feb. 1960, Vol. 13, No. 1, p. 34-36.

149 patients were clinically judged to be Stage I. Irrespective of what was found at surgery or by the pathologist, this stage was not changed. All carcinoma-in-situ were excluded.

These patients were followed for at least five years. Eleven cases had pre-op radiation and subsequent surgery showed no residual cancer, this group had 100% survival. Superficially infiltrating or lesions up to 1 cm. in size were all alive. Lesions that were larger than 1 cm. up to those involving all of the cervix or endocervix but negative nodes showed up to 87% survival. Once the pelvic nodes showed tumor the survival rate dropped to 50%.

Surgery can offer an 87% five year salvage rate for carcinoma of the cervix, irrespective of local size, so long as there are no positive lymph nodes. Clinical staging in 149 patients as Stage I revealed 22 cases or 15% to have pelvic node metastases. Surgical mortality consisted of one case or 0.7%.

F. J. Lone.

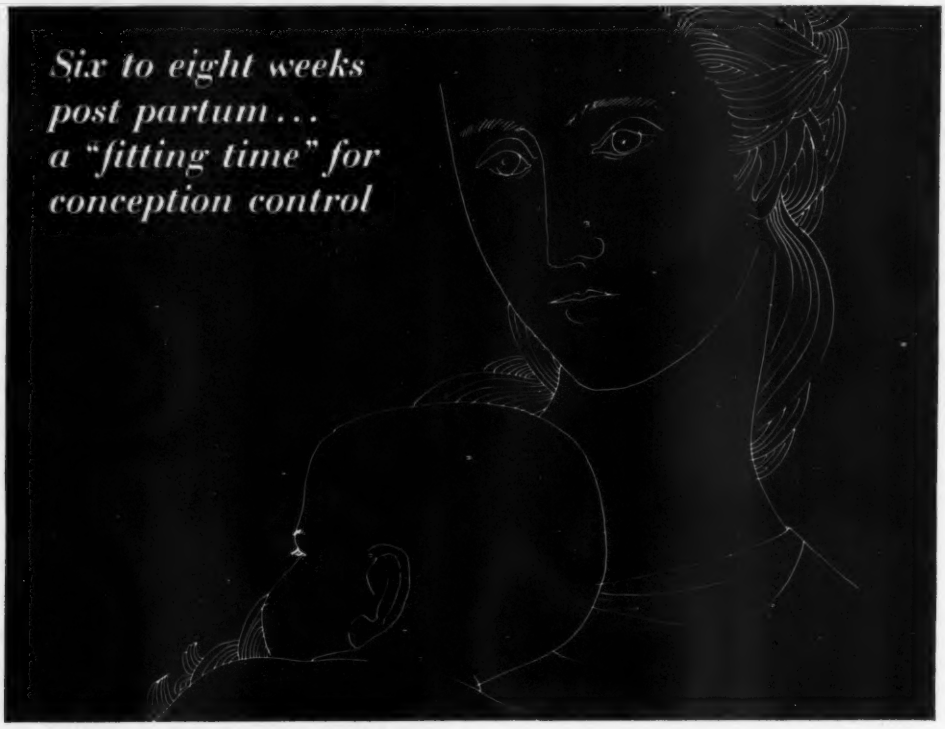
Pulmonary Changes in Hydantoin Therapy. Moore, M. T. J.A.M.A., 171: 1328, 1959.

Hydantoin derivatives used in the therapy of epilepsy may cause such side effects as skin rash, gingival hypertrophy, blood dyscrasia, skin necrosis and a wide variety of neurological changes. The author reports a case of "mottled infiltration throughout the lung fields" occurring in a woman who had been treated with Mesantoin for five years. Extensive diagnostic studies were negative, and the author concluded that the patient had pulmonary fibrosis secondary to hydantoin drugs. Lung biopsy was not performed.

The author then studied chest x-rays on 31 patients selected "at random" from a larger group of patients who were receiving a hydantoin preparation and 27 had roentgenologic changes varying from mild to severe in the form of "accentuated bronchovascular markings" or "diffuse pulmonary fibrosis." Lung biopsy was not performed in any instance, and it is not stated whether diagnostic studies to rule out the presence of any other pulmonary disease were performed. The author notes "an inverse relation between the occurrence of pulmonary fibrosis and gingival hyperplasia." The accompanying reproductions of chest roentgenograms are not convincing.

It is the abstracter's opinion that this is a very uncritical study, and that the thesis that hydantoin therapy leads to any pulmonary changes, much less pulmonary fibrosis, remains to be proved.

G. A. Lillington.



Conception control becomes a matter of special concern six to eight weeks post partum, when the new mother looks to you for advice on the best way to plan the balance of her family. Reliable conception control can be virtually assured with the diaphragm and jelly method, at least 98 per cent effective.[1]

Now—cushioned comfort ... two ways

Your patient experiences special physical comfort when you prescribe either the standard RAMSES* Diaphragm or the new RAMSES BENDEX,* an arc-ing type diaphragm.

The regular RAMSES Diaphragm, suitable for most women, is made of pure gum rubber, with a dome that is unusually light and velvet smooth. The rim, encased in soft rubber, is flexible in all planes permitting complete freedom of motion.

For those women who prefer or require an arcing type diaphragm, the new RAMSES BENDEX embodies all of the superior features of the conventional RAMSES Diaphragm, *together with the very best hinge mechanism contained in any arcing diaphragm.* It thus affords lateral flexibility to supply the proper degree of spring tension without discomfort.

For added protection—RAMSES "10-Hour" Vaginal Jelly†

RAMSES Jelly is uniquely suited for use with either type of RAMSES Diaphragm. It is by design not static, but flows freely over the rim and surface of the diaphragm to add lubrication and to form a spermtight seal over the cervix, which is maintained for *ten full hours* after insertion. It is nonirritating and nontoxic.

You can now prescribe a complete unit for either type of diaphragm. RAMSES "TUK-A-WAY"* Kit #701 contains the regular RAMSES Diaphragm with introducer and a 3-ounce tube of RAMSES Jelly; RAMSES "TUK-A-WAY" Kit #703 contains the RAMSES BENDEX Diaphragm and Jelly tube. Each kit is supplied in an attractive plastic zippered case, beautifully finished inside and out. Both types are now available at key prescription pharmacies.

Reference: 1. Tietze, C.: Proceedings, Third International Conference Planned Parenthood, 1953.

*Trade-marks

†Active agent, dodecaethyleneglycol monolaurate 5%, in a base of long-lasting barrier effectiveness.

*Ramses** *Diaphragms and Jelly*

JULIUS SCHMID (Canada) Ltd., 32 Bermondsey Road, Toronto 16, Canada

Renal Patterns in Myeloma. L. M. Sanchez, C. A. Domz. Ann. Int. Med., Jan., 1960, Vol. 52, No. 1. P. 44-54.

Multiple myeloma may manifest itself with only symptoms and signs of renal disease. Renal insufficiency in the presence of a normal blood pressure should prompt a search for myeloma.

The typical "myeloma" kidney shows marked protein deposition causing tubular obstruction and destruction. There is an associated interstitial reaction which can evoke giant cells.

Paramyloidosis is also found in some cases. The cases present with hypertension.

In spite of high globulin in the serum, many will have a hypogammaglobulinemia. The myeloma protein resembles the normal gamma globulin in shape, size and hydration but is different in its amino-acid structure and in immunological response. Thus, these patients, in spite of high globulin, have a low capacity of antibody response and thus are very susceptible to infection.

Bence Jones protein is specific for myeloma and occurs in every patient. Present clinical techniques are relatively insensitive, showing positive in from 10-50%.

Almost any renal syndrome can be produced by multiple myeloma.

F. J. Lone.

Leiomyosarcoma of the Stomach. John Berg and Gordon McNeer. Cancer, Jan.-Feb., 1960, Vol. 13, No. 1, p. 25-33.

Leiomyosarcoma of the stomach is a rare tumor. At Memorial Centre, New York, from 1935-55 there were 1623 patients with carcinoma of stomach, 24 leiomyosarcoma, 38 primary lymphomas. Analyzing these 24 cases an absolute five year survival rate was 41.7%. The patients varied from 33 to 70 years and there was no sex predilection.

The early lesions for the most part were asymptomatic, or at most vague abdominal complaints. Pain and bleeding were not major complaints.

X-ray diagnoses were very accurate, particularly of the intramural type. The pathology of the tumor determined the prognosis. Size and local extension was most important. Seven of the cases showed mucosal ulceration. The tumors seemed to arise intramurally and extend outside of the stomach, invading local structures.

Histologically, the most important criteria were the nuclei, size, shape and chromatin distribution. Mitotic figures indicated a higher malignancy. Many showed degenerative changes and unusual patterns; these had no relationship to prognosis.

Assessment of local extension (if no metastases noted) was most important to decide curability. Consequently, a frozen section need only state if the tumor is mesenchymal regardless if benign or malignant, since cure depended upon the complete removal of all gross tumour. Of the 24 patients, three were recurrent tumors. Another two were deemed clinically inoperable. The remainder, 19 cases, were explored and three (16%) proved to be inoperable.

Of the remaining 16 cases (76.9% of the original 24 cases) all had gastric resection with hope of cure.

10 cases alive and free of the disease in 5 years. (47.6% of 21 patients)
2 cases died of other causes
1 case lost to follow-up
3 cases died of disease.

Of the 13 determinate patients, 77% were alive and free of disease five years post-operative.

Because other mesenchymal tumors are very rare (including neurilemoma, myoma, etc.) all such tumors should be regarded as leiomyosarcomas and treated accordingly.

F. J. Lone.

The Coexistence of Primary Lung Cancer and Other Primary Malignant Neoplasms. Moertel, C. G., Andersen, H. A. and Baggenstoss, A. H.: Diseases of the Chest. 35: 343, April, 1959.

A solitary pulmonary lesion in a patient having a proved primary malignant neoplasm in another site presents a crucial problem in diagnosis and management. Many authorities feel that a heroic attempt to remove a pulmonary metastatic lesion by thoracic operation is an unjustifiably radical procedure, while others employ such a practice in selected cases with occasional apparent cures. The possibility that the presumed metastatic lesion may in fact be a second and potentially curable primary malignant lesion must be kept in mind. The clinical differentiation between these two situations is difficult and often impossible.

In a group of 1588 cases of pathologically proved primary lung cancer seen at the Mayo Clinic in a ten year period, 65 patients (4.1%) had an independent primary malignant neoplasm at some other site. In almost half the cases, the two primary lesions were diagnosed simultaneously, and in almost all the remainder the diagnosis of the pulmonary neoplasm followed the diagnosis of the other neoplasm by an average of 9.6 years. The distribution of the specific types of associated primary malignant neoplasms did not differ significantly from that expected in a comparable group of patients with single malignant neoplasms.

The authors conclude that the coexistence of primary cancer of the lung and other primary malignant neoplasms occurs with sufficient frequency that no single pulmonary lesion may be assumed to be metastatic without positive pathologic confirmation. In cases affording reasonable evidence that a primary malignancy elsewhere has been controlled, the presence of a discrete pulmonary lesion is an absolute indication for thoracotomy.

G. A. Lillington.

Schering

HORMONES

are minutely standardized, uniformly controlled and biologically adjusted — in-practice-proven assurance of the results you want at reasonable cost to your patients.

on your prescription only

Combined estrogen-androgen — additive relief for menopausal women
GYNETONE Repetabs (ethinyl estradiol with methyltestosterone)
GYNETONE injection (estradiol benzoate with testosterone propionate)

Specific androgen therapy—for the elderly and in the male climacteric
ORETON (testosterone) oral, injection, pellets

Estrogens — for the female climacteric
ESTINYL (ethinyl estradiol) tablets

Progestins—in habitual abortion, dysmenorrhea and premenstrual tension
PRANONE (ethisterone) tablets
PROLUTON (progesterone) injection, buccal tablets

Schering
CORPORATION LIMITED MONTREAL

Ocular Manifestations of Pituitary Tumor in Cushing's Syndrome: Kearns, T. P., Salassa, R. M., Kernohan, J. W. and MacCarty, C. S. A.M.A. Arch. Ophth. 62: 242-247, (August), 1959.

In a series of 158 patients treated by adrenal resection for Cushing's syndrome, adrenal cortical tumor was present in 34 cases, and roentgenographic examination of the skull failed to show evidence of a pituitary tumor in every instance. In the 122 patients with Cushing's syndrome due to adrenal hyperplasia, there was roentgenographic evidence of a pituitary tumor either before or after adrenalectomy in 12 cases. Ocular symptoms were present in six of these 12 patients; five of this group had visual field defects of a chiasmal nature, and four of the six patients had palsy of the third cranial nerve. The onset of visual symptoms was usually rapid and the field defects were not of the classical pattern commonly associated with pituitary tumor.

Histological study of the pituitary tumors was possible in all six patients, the tissue being obtained at craniotomy in five and at necropsy in one. In all instances, the tumors were chromophobe adenomas. One tumor was highly malignant, with metastases in the spinal cord and the liver. In no instance was a basophilic adenoma present.

The authors recommend that roentgenographic study of the sella and ophthalmoscopic examination should be routine in the initial examination and subsequent evaluation of patients who have Cushing's syndrome. Pituitary tumors occur or develop subsequently in 10% of such patients, and the tumors appear to be more active in growth characteristics than the usual pituitary tumor. It is suggested that relief of Cushing's syndrome by total adrenalectomy actually may result in the growth of pituitary tumors.

G. A. Lillington.

for RAPID and PROLONGED CONTROL of HYPERACIDITY

"ALAMINO" COMPOUND

Aluminum glycinate, basic*..... 7.7 gr. (0.5 G.)
Atropine sulphate..... 1/500 gr. (0.13 mg.)
Butabarbital NND..... ¼ gr. (16 mg.)

DOSAGE: *One tablet before each meal and one or two tablets at bedtime.*

Bottles of 100 tablets.

*Patented, 1951

- *non-absorbable*
- *no danger of alkalosis*
- *prompt relief of pain in uncomplicated peptic ulcer*
- *gastric contents buffered to a pH at which peptic activity is completely inhibited*

Charles E. Frosst & Co.
MONTREAL CANADA

when emotional turbulence threatens medical or surgical care

Fear, agitation, and resistance often hinder medical diagnosis and treatment.

SPARINE alleviates agitation, overcomes resistance, placates fears.

In addition to calming the patient, SPARINE controls other interfering symptoms: nausea, vomiting, and hiccoughs.

Sparine

HYDROCHLORIDE

Promazine Hydrochloride, Wyeth

INJECTION • TABLETS • SYRUP

Wyeth

Reg. Trade Mark

WALKERVILLE, ONTARIO

Social News

Reported by Marjorie R. Bennett, M.D.

Our Katy is under the weather as we go to press. Reports of her progress are encouraging and we hope she will be in circulation very soon. Good luck, Kay!

☆

Dr. Max Desmarais, who left Winnipeg in January for Morocco to direct a medical team working among victims of paralysis due to the use of adulterated cooking oil, is now heading Red Cross emergency operations in the earthquake stricken city of Agadir. Dr. Desmarais is a director of physical medicine at Winnipeg Municipal, St. Boniface and Misericordia hospitals. He will return here to practice in the summer.

☆

Dr. A. J. W. Alcock is attending a course in San Francisco on the physiological aspects of diagnosis and treatment. Following the course, which is organized by the American Physiological Society, Dr. Alcock will visit hospitals in the San Francisco area.

☆

Our congratulations to Dr. Charles H. Hollenberg, who is one of four Canadians appointed this year as Markle scholars in medical science. Dr. Hollenberg is the son of the late Dr. Abraham Hollenberg, and is now a research fellow at New England Centre Hospital, Boston. He will become a lecturer at McGill University in Montreal in July.

☆

Dr. A. H. Neufeld of Montreal has been named professor and head of the Department of Pathological Chemistry at the University of Western Ontario. Dr. Neufeld is a graduate of the University of Manitoba.

☆

Dr. D. Lewkin, psychiatrist and director of the Mental Health Clinic at Port Arthur, visited Fort Frances recently to confer with district physicians and health officials. Dr. Lewkin was representing the Ontario Department of Health, which is introducing mental health services for the area.

Miss Jane Hook of Kenora, Ont. and Dr. J. Hayakawa were married in Our Lady of Victory Church on February 27, 1960.

☆

The stork, as you will see, has been working diligently. We record the following visits:

To the home of Dr. and Mrs. D. Allen of Flin Flon, where he left a son, Scott Randall.

☆

To Dr. and Mrs. Gordon Steenson of Ashern, Man., a son, Donald Edward, a brother for Bob, Valerie, Janice and David.

☆

To Dr. and Mrs. C. J. Mongeon of Sioux Falls, S.D., a son, Christopher Phillip.

☆

To Dr. and Mrs. Robert Lee, a son, William Musgrove.

☆

To. Dr. and Mrs. Colin Sinclair, a daughter, Carolyn Marie.

☆

To Dr. and Mrs. W. May, a daughter, Myra Elizabeth.

☆

To. Dr. and Mrs. Russell Dunlop, a son Normar John, brother for Robbie and Susan.

for these two conditions met almost daily in general practice

MILD AND SEVERE NAUSEA AND VOMITING
MILD AND MODERATE MENTAL AND EMOTIONAL DISTURBANCES

Stemetil
PROCLORPERAZINE

POTENT ANTIEMETIC NEUROSTATIC
specific action on the neuro-vegetative system
effective in low dosage potent and rapid action
freedom from drowsiness and depressing effects

TABLETS
SUPPOSITORIES
AMPOULES
MULTIDOSE VIALS
LIQUID 5 MG.
LIQUID 15 MG.

Poulenc LTD.
8580 Esplanade, Montreal

Study Committee on Services to Unmarried Parents

The Doctor and the Unmarried Mother

In the Province of Manitoba in 1958, there were 1,098 children born out of wedlock—853 were white children and 245 were Indian.

In the same year 689 new unmarried mothers came, or were referred, to the four Children's Aid Societies, the Jewish Child and Family Agency, and the Rural and District Offices of the Department of Public Welfare for help in planning for themselves and for their babies. At January 1, 1958, there were 729 unmarried mothers already known to the above agencies making a total of 1,418 who received counselling and help in 1958.

The earlier an unmarried mother is referred to the social worker, the more helpful is the service given her toward constructive planning for herself and her baby. The social worker is prepared to help an unmarried mother in many ways, making practical plans for living arrangements until her confinement; contacting the putative father regarding his responsibility and possible financial assistance; giving advice regarding legal counsel when necessary; making the arrangements for medical care; exploring plans for herself and the baby. Above all, in many instances, the social worker helps the mother understand her own deep feelings about her condition.

Many unmarried mothers are ill-prepared or completely unable to make realistic plans. They are hostile, frightened, withdrawn and have numberless questions to which they desire answers so they may face this experience realistically. The special training and skills of the social worker are available to these girls. For instance, if adoption for the baby is the mother's choice, the legal and social procedures necessary to place a child in a home are undertaken by the social worker who knows these procedures.

The doctor is frequently the first person to know of the unmarried mother's condition. He may find he is unable to answer her many questions about where she should go for care, and what plans she might consider for her baby. The Children's Aid Society or District Welfare Office in the area in which the girl resides would be pleased to have her referred as soon as possible. The social worker would gladly discuss an alternative plan with the doctor where the unmarried mother does not wish to be referred to her local welfare agency.

Social Agencies are finding that, with the Hospital Services Plan, many unmarried mothers are not known to them until they are referred by the hospital following confinement, when the mother wishes to leave hospital without the baby. Planning with the unmarried mother is extremely difficult at this stage, and interim care for the baby is often necessary until the mother can make up her mind

LEUKORRHEA

VAGINAL INFECTIONS

CONES AND TABLETS

TWO FORMULAS

OVOQUINOL-PLAIN

	Cones	Tab.
Diiodohydroxyquinoline U.S.P.	75 mg.	75 mg.
Sodium Propionate	500 mg.	250 mg.
Sulfadiazine U.S.P.	400 mg.	400 mg.
Phenoxyethanol B.P.C.	0.04 ml.	
Dextrose and Lactose	q.s.	q.s.

OVOQUINOL-OESTRO

Same formula as OVOQUINOL-PLAIN plus 0.01 mg. Ethinyl-oestradiol B.P. per cone or tablet.

DOSAGE

One or two cones or tablets per day preferably at bed-time or as prescribed by the physician.

NADEAU LABORATORY LTD.

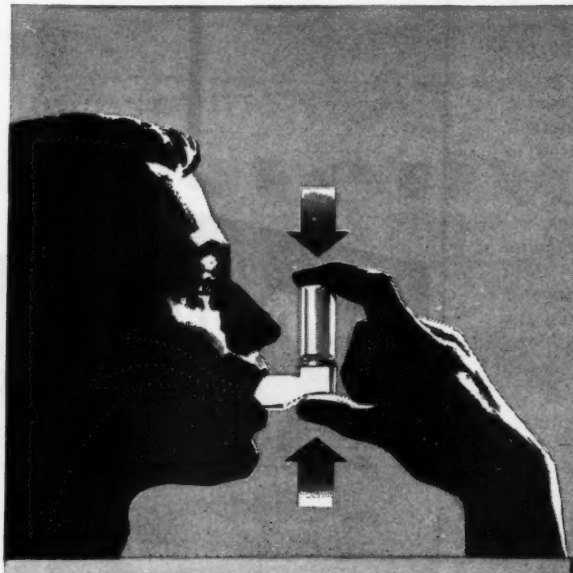
Montreal Canada

In Asthmatic Attacks...

AMPLE AIR IMMEDIATELY

with Medihaler®

automatically measured-dose aerosol medications



- Ready and in use in *5 seconds* under any circumstance.
- Travels with the patient anywhere...Can be concealed in the hand... Can be carried in vest pocket or purse.
- Dose is metered and medication is propelled automatically with single-stroke finger pressure. 200 doses per vial.

Prescribe either of two bronchodilators: isoproterenol or epinephrine

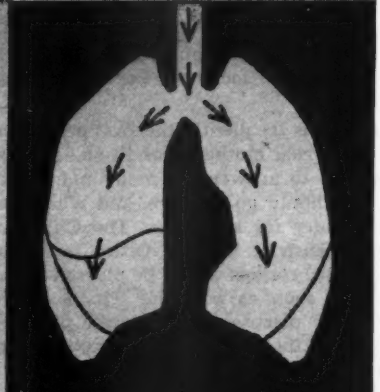
Medihaler-ISO®*

Isoproterenol sulfate, 2.0 mg. per cc., suspended in inert, nontoxic aerosol vehicle. Contains no alcohol. Each measured dose contains 0.06 mg. isoproterenol.

Medihaler-EPI®*

Epinephrine bitartrate, 7.0 mg. per cc., suspended in inert, nontoxic aerosol vehicle. Contains no alcohol. Each measured dose contains 0.15 mg. epinephrine.

***First Rx: vial of medication with oral adapter**
Repeat Rx: can specify refill vial only



22½% greater vital capacity within seconds after inhalation... medications premicronized to particle size which assures fastest delivery to alveolar spaces.

Riker
COOKSVILLE, ONT.

what she wishes to do. It is more beneficial to both mother and baby when this planning can be done during the pre-confinement period.

Rehabilitation for the unmarried mother is one aspect of this problem which is often overlooked. When there has been little or no pre-confinement contact, it is often impossible to work out a plan. When a social worker is able to form an earlier relationship with the unmarried mother, rehabilitation can be a successful part of the total planning.

Pamphlets which answer some of the most common questions raised by the unmarried mothers are available to doctors and anyone interested. These pamphlets also provide the names and addresses of the different social work agencies and departments in the province to which the unmarried mother can be referred for help.

Miss Winona Armitage,
Study Committee on Services to
Unmarried Parents.

☆

Physicians' Art Salon at C.M.A. Convention Invites Exhibitors, June 13-17 Banff, Alberta

The Physicians' Art Salon Committee cordially invites Canadian physicians and medical undergraduates to enter paintings, photographs or colour slides in the 1960 Salon to be held in the Banff Springs Hotel, from June 13th to 17th. This will mark the 16th year that this interesting art and photographic feature will be held at the annual C.M.A. Convention. It is again sponsored by Frank W. Horner Limited, Montreal, Quebec.

Conditions of Entry

Entries will be accepted in three sections:

1. Fine Art
2. Monochrome Photography
3. Colour Photography.

The Fine Art section is further subdivided into three categories: Traditional, Contemporary (Modern), and Portrait. Classification into these categories is done by the judges. There is no restriction on media: oil, tempera, gouache, water colour, charcoal, pencil, or dry brush is acceptable in each.

Each exhibitor may submit up to three entries in the Fine Art and Colour Photography and four in Monochrome Photography. Exhibitors may enter up to the limit in one or more sections. There is no charge. All costs, including transportation to and from Banff, will be borne by Horner.

Judging and Awards

All accepted entries will be displayed in the Salon and then judged for awards by a competent jury selected by the Art Salon Committee.

To obtain entry form

Any physician or medical undergraduate may obtain an entry form and complete details from the sponsor at P.O. Box 959, Montreal, Quebec. A short note or post card will bring the form along with complete instructions on how to prepare and ship your entries.

Art Salon Calendar

The Physicians' Art Salon Calendar, an attractive desk piece based on Salon exhibits, will again be prepared by Frank W. Horner Limited. The Calendar reproduces selections from the award winners and is distributed to all physicians in Canada with the compliments of the Company.

☆

The Neil John Maclean Memorial Award for Clinical Investigation

Conditions governing the Award:

1. Applicants must be graduates in Medicine of not more than five years, residing in Manitoba. The adjudicating Committee, however may vary this condition so as not to exclude internes or fellows who have been engaged in general medical service with the Forces or in civilian practice.

2. Residents or internes of teaching hospitals should submit their papers through the Chief or a senior member of the Department in which the work is being carried out. If an applicant is not on the staff of a teaching hospital he should apply directly to the Honorary Secretary.

3. The submitted paper must be based on observations made in large part by the applicant himself while a resident of Manitoba. Credit will be given for evidence of independent thought.

4. The paper should be written in the style used in an appropriate medical journal.

5. The paper must be entirely written by the applicant but in its composition he is free to obtain any advice or criticism which he may desire. Preference will be given to papers with a bearing on medical practice.

6. The applicant may be required to appear in person before the adjudicating Committee.

7. Assignment of the award shall be decided by the Awards Committee of the Winnipeg Clinic Research Institute. Members of the Committee will read the submissions and list them in order of preference. The Committee or those delegated by the Committee will make the final decision.

8. Submissions should be in the hands of the Honorary Secretary, Winnipeg Clinic Research Institute, 205 Vaughan Street at St. Mary's Avenue, Winnipeg 1, Canada by April 15th.

9. The award will be available annually and will consist of a prize of $250.00, plus a suitably engraved certificate.

When your patient needs

nutritional support

plus

mild sedation

BĒPLETE

VITAMIN B FACTORS WITH PHENOBARBITAL, WYETH

ELIXIR • TABLETS

Each 5 cc. teaspoonful contains:

Phenobarbital	0.25 gr.
Thiamine	1 mg.
Riboflavin	1 mg.
Pyridoxine	0.4 mg.
Vitamin B_{12}	1.67 mcg.
Niacinamide	10 mg.
d-Pantothenyl Alcohol	2 mg.

Restoration of a normal emotional picture is often facilitated by including BĒPLETE in the therapeutic regimen

SUPPLIED:

ELIXIR: Bottles of 16 fl. oz. and ½ Imp. gal.
TABLETS: Bottles of 100 and 1000.

Wyeth

Reg. Trade Mark
WALKERVILLE, ONTARIO
MONCTON MONTREAL WINNIPEG VANCOUVER

Association Page

Reported by M. T. Macfarland, M.D.

The Association announces the appointment of Mr. Richard P. H. Sprague as Assistant Executive Director effective March 1, 1960. Mr. Sprague brings to his new appointment several years experience of an executive capacity in a related field, having most recently been associated with the Manitoba Hospital Services Plan since its inception and prior to that for nine years Hospital Relations and Claims Manager for the Manitoba Blue Cross Plan.

Mr. Sprague was born and educated in Winnipeg and both prior to and immediately following the last war was with a large financial and insurance organization for a period of nine years. He served in the army during the war with the Royal Winnipeg Rifles, transferring to the Durham Light Infantry of the British Army for the Northwest Europe Campaign.

Committee Activities

It has been suggested that the membership-at-large would like to know something of the activities of the various committees of the Association. On the whole all committees appear to have been very active during the past few weeks and have dealt with many and varied subjects.

Public Relations Committee

It may not be generally known that Mr. Gene Telpner of the Winnipeg Free Press has been appointed on a part time basis as a press relations officer for the Association. It was decided that Mr. Telpner would produce specific articles about the Profession that would be of interest to the general public. Mr. Telpner is working on material at the present for release in the near future.

The committee spent a lot of time and effort on the Citizens' Forum Program and it is reported that a film on the Eye Bank Program will be a very interesting one and there is a possibility that it will be used on a National T.V. hook-up. Speaking about T.V. it may be of interest to note that a National program under the title of "A Disordered Mind" will be seen at 10:30 p.m., Wednesdays on the following dates: April 20, April 27, May 4 and May 11.

The committee has been interested in the Association's Conciliation Committee and the best means of publicizing the existence of such a committee in order that the general public may be familiar with the means at their disposal to resolve problems they may have with the Profession. At the present time patients' problems are referred to the Executive Director and the matter resolved with assistance from the Officers if required.

The Association has recently purchased a documentary film about the National Health Service in Great Britain entitled "On Call to a Nation." This film is expected to arrive very shortly and it will be available for showing.

The committee chairman will attend a C.M.A. Public Relations meeting during the first week of April and will be reporting upon the activities of his local committee and will in turn bring back information of activities on a National level.

The Annual Meeting Committee and the Scientific Program Committee

The Annual Meeting Committee and the Scientific Program Committee have had several meetings in connection with the Annual Meeting set for September 26th and 27th at the Marlborough Hotel.

Committee members have now been named to plan the many phases of programming, etc. and from tentative arrangements now made in respect to accommodation, speakers, and scientific exhibits it appears that the Annual Meeting will once again be a first rate affair. It is to be noted that immediately following the M.M.A. Annual Meeting will be a two day meeting of the International College of Surgeons. It is expected that the I.C.S. meeting will draw many persons from the States and Canada.

Economics Committee

The Economics Committee has spent considerable time in connection with the proposed indigent care program. Although it is a little early to make a definite statement at the present time it can be reported that progress is being made in reaching a reasonable working arrangement with the Provincial Government.

The committee wishes to announce that an interesting series of lectures will be given to fourth year students on April 9th, 23rd and 30th. The subjects will include discussion on matters pertaining to legal and accounting problems in setting up practice and also information on prepaid medical plans.

The Committee on Medical Aspects of Traffic Accidents

The committee finds that this is a very broad subject with far reaching implications. Recently the committee has been concerned with such subjects as the care of injured patients at the scene of a highway traffic accident, the transportation from the scene of the accident to the site of treatment, and a method of education for the public regarding the increased chance of injury in improperly designed automobiles and in passenger cars not equipped with safety belts.

The committee has been approached by the Motor Vehicle Branch with a view to setting up a medical consulting board. Progress is being made in this respect. Committee members are awaiting material from the B.C. Medical Association and from the Center for Safety Education at New York University concerning requirements for a drivers license and design of a medical certificate. A suggested

saturation doses – the hard way!

Each of these food portions contains a saturation dose of one of the water-soluble B vitamins or C. The easy way to provide such quantities of these vitamins with speed, safety and economy is to prescribe Allbee with C. Recommended in pregnancy, deficiency states, digestive dysfunction and convalescence.

In each Allbee with C:		As much as:*
Thiamine mononitrate (B_1)	15 mg.	6.9 lbs. of fried bacon
Riboflavin (B_2)	10 mg.	31½ ozs. of liverwurst
Pyridoxine HCl (B_4)	5 mg.	2 lbs. of yellow corn
Nicotinamide	50 mg.	11 ozs. of roasted peanuts
Calcium pantothenate	10 mg.	¼ lb. of fried beef liver
Ascorbic acid (Vitamin C)	250 mg.	⅔ lb. of cooked broccoli

*These common foods are among the richest sources of B vitamins and ascorbic acid. H. A. Wooster, Jr., Nutritional Data, 2nd Ed., Pittsburgh, 1954.

Allbee® with C

Robins

A. H. ROBINS CO. OF CANADA, LTD.
MONTREAL, QUEBEC.

medical certificate was drafted by the Motor Vehicle Branch for consideration of the Committee.

It appears that much information on the education for Accident prevention may be obtained from the Indiana State Police. The committee is exploring ways and means of using this material.

The chairman of the committee reported on the C.M.A. National Committee Meeting held in March at Ottawa. The main topics discussed were as follows: 1, the prevention of Traffic Accidents; 2, Transportation and communication following a traffic accident; 3, emergency care of injuries at the hospital.

Constitution and By-laws

The committee recently met to review several suggested changes to tidy up the by-laws. The points for consideration were the election of senior members, and the manner of appointment of the Professional Policy Committee. The committee recommended that the election of senior members be by an indicative mark on the ballot and are giving further thought to the Professional Policy Committee.

Terms of reference were determined for the Medical Aspects of Traffic Accidents Committee and the Workmen's Compensation Board—Medical Board of Reference.

The committee pointed out that the name of the Manitoba Health Service where it appeared in the By-laws be now corrected to Manitoba Medical Service to be consistent with the change requested by Legislation.

According to the By-laws of the Association all amendments to the By-laws must be ratified at an Annual Meeting.

Hospital Relations Committee

It may not be generally known that a joint committee on Hospital Relations has been formed with the Associated Hospitals of Manitoba. The purpose of the Joint Committee is, of course, to discuss subjects or problems of mutual interest to the two bodies. At a recent meeting of the Joint Committee the matter of establishing Tissue Committees was discussed. The proposed Government regulation for the examination of tissues removed at operations in hospitals in Manitoba was reviewed.

This subject was also on the agenda at a recent meeting of the Hospital Relations Committee at which Dr. Wood of the Standards Division of the M.H.S.P. was an invited guest. The matter of establishing Tissue Committees is still under discussion by the committee.

The Hospital Relations Committee will shortly tackle another problem—the review of suggested Hospital By-laws as drafted by the Associated Hospitals of Manitoba. It appears that the M.H.S.P. Standards Division is also interested in this subject.

Finance Committee

It was budget time for this committee at their recent meeting. Although expenses appear to be at an all time high, revenue is expected to keep pace with expenditures and this coupled with another financially good year for the Review should see the Association end their year at the break-even point. It was encouraging to learn that the Honorary Treasurer did not propose an increase in the Association's fees for the current year.

THE ANNUAL MEETING

OF THE

MANITOBA MEDICAL ASSOCIATION

(Canadian Medical Association, Manitoba Division)

will be held on

SEPTEMBER 26th and 27th, 1960

MARLBOROUGH HOTEL, WINNIPEG

THE FIRST
OF THE NEW PENICILLINS

BROCSIL

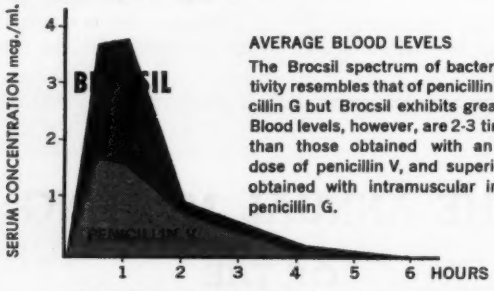
BRL 152
The potassium salt of 6-(ω—phenoxy-propionamido)—penicillanic acid

An oral penicillin more effective than parenteral penicillin G

Brocsil is the first of the new antibiotics prepared from the penicillin "nucleus"—6-aminopenicillanic acid—originally isolated in the Beecham Research Laboratories. It exhibits considerable advantages over all kinds of penicillin.

Brocsil produces greater antibiotic activity in the blood following oral administration than has ever before been achieved with penicillins—twice as great as that of penicillin V.

Blood levels after oral doses are superior to those following intramuscular injections of penicillin G.



AVERAGE BLOOD LEVELS

The Brocsil spectrum of bacteriological activity resembles that of penicillin V and penicillin G but Brocsil exhibits greater activity. Blood levels, however, are 2-3 times greater than those obtained with an equivalent dose of penicillin V, and superior to those obtained with intramuscular injections of penicillin G.

Chart shows blood levels obtained in studies following 250 mg. of Brocsil and Penicillin V orally.

Recommended dosage 125 mg. to 250 mg. t.i.d. according to severity of infection. Information on request.

BROCSIL IS NOW AVAILABLE IN 125 MG. (200,000 I.U.) tablets
250 MG. (400,000 I.U.) tablets

BEECHAM RESEARCH Laboratories Ltd.
P.O. Box 99, Weston, Ontario.
A new scientific pharmaceutical research organization into which C. L. Bencard has been integrated.